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TROPHIC GLIAL STATES IN SPINAL CORD LESIONS.[1]

By OLIVER LATHAM, M.B. (Sydney),
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THE supporting glial tissue of the spinal cord is normally a strong resiliant network which ably performs its function of providing a support for the neurones and blood vessels of the part. In practice this can be readily noted in that histologists can not only cut by hand sections unsupported by any embedding material as thin as ten microns, but also subject these with impunity to the action of mordanting, staining and clearing reagents. Its fibrillar nature is manifest and this is greatly proliferated in disseminated sclerosis *et cetera*.

Now as it acts the part of an interstitial tissue, one would expect it to outlast the neurones when in various spinal lesions the latter sicken and die. And this is so. We find that in many forms of spinal degeneration the glia actually proliferates and fills up the empty spaces. Sections can still be cut and manipulated with impunity. However, one occasionally comes across cords wherein the glia has no such properties. In these cases it looks more dense, has more or less lost its fibrous nature, it has become so granular and brittle that satisfactory sections can be got only by embedding in celloidin. Sometimes too it merges with the pia-arachnoid which may then share this state. Large or small lengths of the cord may be thus affected, while very commonly the medulla and parts above quite escape. In our own practice we have found that this is most likely to obtain in conditions allied to compression myelitis (more fully discussed later), whose apparent cause is far removed from the site of the lesion. In these diseased states the glial atrophy or degeneration may be out of all proportion to the accompanying axonal degeneration in the neurones. As I have recently been handed for examination the brain and spinal cord from an obscure case of Addison's disease (a full clinical and pathological account of which will possibly be published later) exhibiting not only those spinal lesions included under the term "the combined scleroses," but also this atrophic condition of the neuroglia, I thought it would be of some interest to review some previous cords manifesting a like state.

Briefly examination of this specimen revealed many cerebral pontine and spinal neurones undergoing axonal degeneration, but save for a slight paling of the cerebellar tracts in the medulla the

[1] Read at a meeting of the Sections of Neurology and Psychiatry and Pathology of the New South Wales Branch of the British Medical Association on October 15, 1925.

only gross lesions noted existed in the cord, where Weigert-Pal methods showed widespread macroscopic degeneration in the posterior columns, cortico-spinal and cerebellar tracts and the above-mentioned glial atrophy. No degeneration was present in the root zones, anterior or posterior nerve roots or peripheral nerves.

If in this case the glial atrophy is due to the same underlying cause which brings about the neuronic degeneration, one may ask why the glia more often proliferates than degenerates in myelogenous lesions. In these combined scleroses some would incriminate a diseased state of the blood acting on the part through long narrow tortuous blood vessels.[1] However, the glia commonly maintains its nutrition as also it does in tabes, cerebral lesions and quite large tumours of the cord, especially when they are of a gliomatous, endotheliomatous or angiomatous nature. Tumours of the more frankly carcinomatous and sarcoma type seem far more destructive to glia. Simple pressure will not account for the phenomena, but the fact that a sharp tap[2] on some overlying bone may cause a steady progressive myelitis of the "primary non-reactive degenerative type" when no internal injury can be detected, made me think of a sort of "shock" effect accidentally injuring the "trophic controls" of the parts, however such control existed.

If we could assume the presence of trophic nerves (running perhaps in the sympathetics) to the several structures composing the spinal cord, we might find it much easier to understand how the degeneration is more evident at one time in the supporting structures, at others in the neurones. The neurones themselves may be under the control of special trophic nerves which may ultimately be proved to be a part of the sympathetic system. Then whatever the ætiology of the spinal lesion (though the more specialized neurone would more often be the chief sufferer) the actual incidence of the degeneration would fall on that tissue whose trophic nerves were most affected.

Some very interesting work has been done lately on the subject of special fibres included in the sympathetic system directly concerned with the nutrition of the part quite apart from vasomotor nerves. This work may perhaps decide such factors as (i.) the maintenance of the organ or health of the neurones, (ii.) whether it will undergo repair in disease, fail to be repaired or become over repaired as in hypertrophy of keloid,[13] (iii.) or whether apart from apparent disease or injury the cell unit will from no apparent cause commence to hypertrophy or slowly or in some cases rapidly atrophy and die, often in the midst of apparently healthy units and with the minimum accompanying evidence of inflammation or reaction.

A *résumé* of some observations on degenerative lesions associated with different causes may throw some light on these nervous phenomena. It will be noted that a neurone may in dimensions reach far beyond microscopical size and that inches of it may be diseased while its cell body and other processes of it appear quite healthy. In "combined sclerosis" commonly the part within the cord gave most evidence of atrophy. We may here note the distinction between "lymphogenous" degeneration (with considerable reaction both without and within the cord) and "hematogenous" degeneration in which most often the main stress of atrophy lies inside the cord. Orr, Rows and Sturrock[3] [5] placed celloidin capsules containing organisms against the sciatic nerve of rabbits and in due course traced chronic inflammatory reaction with lymphocytosis and fibrosis (with degeneration afterwards) under the epineureum of the nerves, posterior spinal ganglion, piaarachnoid and ultimately they found a tabetic-like lesion in the posterior columns. This would be their lymphogenous reaction. In another series these capsules, placed by the side of certain abdominal sympathetic ganglia, led primarily to the same sort of inflammatory reaction in the ganglia; the spinal nerves escaped. Not so the cord, however. It manifested a slowly developing myelin degeneration or atrophy with minimum reaction in piaarachnoid, perivascular sheaths or surrounding glia in the posterior and lateral columns. They distinguished the results by calling the latter a "hematogenous" degeneration and the areas involved resembled those obtaining in the combined scleroses. Another almost similar form of neuronic atrophy wherein certain neurones are picked out, may be seen in the so-called "compression myelitis" studied by Minor[4] in hæmatomyelia, by Williamson[1] in connexion with vertebral caries, by Gordon Holmes[2] in connexion with spinal (including cranial) injuries of warfare and by Paul[6] in connexion with disseminated softenings in the cord in cases of pancreatic cancer. In these cases individual nerve cells manifest axonal degeneration, the nucleus is displaced, the processes are twisted, the axis cylinders are greatly swollen and the myelin sheath is enormously thinned out and dilated before it disappears. Now the interesting point about the compression myelitis in tumours, vertebral caries and these glancing bullet wounds just touching a spinous process or occipital protuberance is that no direct pressure or injury can be detected in the underlying nervous tissue or even connecting the atrophic lesion with the trifling wound or bone disease. In a case of ours[8] the disease was cancerous glands in the neck. These authors suggest some obscure interference with the blood or lymph flow, but surely far more severe bone fractures with even involvement of the nervous tissue itself, not to mention many cases of intramedullary tumours which must have had a real interference with circulatory conditions by pressure alone, have existed without any sign of this myelitis. Nevertheless the supporting neuroglia in these compression myelitic cords, while manifesting but little reaction, has become extraordinarily brittle and granular, all pointing to some influences brought to bear on the trophic enervation of the part or at least on trophic conditions.

Has sufficient attention been directed to possible damage to either sympathetic nerve filament or ganglions in these experiments and experiences?

Some recent physiological work in support of special (sympathetic) nerves for nutrition, hypertrophy, involution and repair may have a bearing on this question. Orbeli[9] holds that striped and cardiac muscle are very closely related and are similarly affected by the sympathetic as regards strength, irritability, conductivity and tone. Ginetsinsky, using isolated frog's gastrocnemius muscle severed from its vascular supply, but with all nerve connexions intact, found that when the muscle had become fatigued by over stimulation of the roots of the eighth and ninth spinal nerves, stimulation of the sympathetic fibres at the level of the seventh ganglion caused a gradual restoration of the function of the greatly fatigued and contracted muscles and this lasted a long time. Japanese observers had noted the same for the heart and all of you are acquainted with Hunter and Royle's[11] work on birds' muscles with cut sympathetics. Orbeli, summing up, says:

> We must broaden our horizon and recognize both skeletal as well as cardiac muscle come under the direct influence of the sympathetic, fulfilling the prophecy of the "chemical tonus" of Pfluger and the "trophic innervation" of Pavlov, in a word the sympathetic must also affect their metabolism.

Pavlov[10] has long sided with the clinicians regarding the existence of trophic nerves and lately has satisfied himself they can be demonstrated in certain organs. He holds that the heart has not only (i.) vasomotor, (ii.) accelerator and inhibitory nerves, but also (iii.) a pair of trophic nerves which control the amount of material used there and indeed in each organ in the body, including, may we add, the neurones and glia of the central nervous system itself!

He also noted that certain skin lesions and injuries were associated with regular visceral phenomena and *vice versa*, intestinal operations with trophic skin lesions. Clinicians, of course, recognize duodenal ulcer after burns and skin rashes in children with intestinal complaints. And again softening of bones occurs in dogs when damp cold is continually applied to the skin. These phenomena could be explained as reflexes from abnormally stimulated centripetal nerves of the alimentary tract to inhibitory nerves to different tissues, one set augmenting, the other diminishing its vitality and if extremely irritated destroying altogether the resistance of the tissues. Shock and trophic phenomena, therefore, are reflexes.

The reflex irritation when it acts quickly on the inhibitory nerves, produces the symptoms of shock, when it acts more slowly and over a long period chronic pathological, that is trophic, changes take place in the tissues. The preservation of the heart and the central nervous system in starvation would argue a special selective augmentary nutritional flow to these organs so necessary for the preservation of the individual. The issues are complicated by our ignorance in many cases. For instance, some forms of the myelites of the Landry class hitherto obscure in ætiology seem more readily grasped when attention is paid to observations such as those of Goodpasture[10] who directly inoculated the *corpus luteum* of pregnant rabbits or the suprarenal with the virus of *herpes simplex*. In twenty-four hours the *corpus luteum* cells held well developed inclusion bodies and eventually an acute myelitis followed reaching the cord at the point where the sympathetic fibres from these organs were connected. From the cornea the brain was reached by the sensory division of the fifth nerve and the lumbar cord from leg muscle infection, transmission being by way of the axis cylinders.

Other clinical evidences bearing on the existence of trophic nerves doubtless occur to you. Such are perforating ulcers and tabetic joints, brittle bones in paralytic dementia, multiple whitlows in encephalitis. Bayliss,[12] always careful, admitted the validity of Campbell's claim for a trophic cause for his blebs when he demonstrated the destructive effect of the virus of herpes on the posterior spinal ganglias supplying the area of the lesion. Again Todd[14] discusses various changes in blood vessels such as hypertrophy and obliteration and changes in nerve lesions. The lower limb of a middle aged male patient was handed us for examination; it was as hard as cartilage; we found that the muscle fibres manifested the changes of pseudohypertrophic muscular dystrophy, the arterioles were almost obliterated by fibro-hyaline material, while the nerves were encased in a dense atrophic fine fibrillar sclerosis. Collier[15] quotes McCarrison as saying that one-third of his endemic cretins suffered from nerve symptoms comparable to Little's disease. In other words, glandular, trophic and nervous conditions were associated in quite a large class of patients. Camus and Roussy describe a condition called "puff ball rats" as eventuating when the suprapituitary region is pricked, the suggestion being that this area is the upper limit of the reticulated substance to which a sympathetic function is ascribed in the midbrain and cord. Chromatolysis[17] has been found in the vegetative nuclei of the ninth and tenth cranial nerves in trophic lesions and Myerson[21] finds in the psychoses considerable damage in certain of the cervical sympathetic ganglia. Recent writers[16] implicate the nervous system in cases of *osteitis deformans* and Boveri[7] implicates the "lizard skin" phenomena in syringomyelia. Trophic disturbances and neuritis are well known in beri beri and Inglis and Bell[20] lately described a case in which hypertrophy of the third digit with cartilaginous growths had occurred associated with a plexiform neuroma of the median nerve.

Experimental physiologists,[19] cutting the anterior and posterior nerve roots to the hind limb, claimed that healing was not interfered with, but then they did not cut the sympathetics! Others who performed similar operations said the sores came from the obvious injury received by paralysed limbs, but such lesions do not follow section of the cord between the dorsal and lumbar region.

From the histologists as a result of the use of the intravitam methylene blue method, further evidence is to hand in the recent discovery of rich terminal arborizations from unmedullated nerve fibres (prob-

ably sympathetic) upon simple fat cells. These terminations were quite distinct and separate from the capillary nerves which were also clearly visible. They were found by Dr. Wilkinson[22] during the course of a systemic survey of the nerve endings in the body while working in conjunction with Professor Burkitt in the Histology Department of the University of Sydney. To recognize them the most critical illumination and highest powers of the microscope are necessary. It is to be hoped that further knowledge gained from such diverse sources on the functions of possible trophic nerves will render easier of interpretation neuronic and interstitial lesions of the spinal cord.

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DIGESTION AND MENTAL DISEASE: AN ANALYSIS OF ONE HUNDRED CONSECUTIVE FRACTIONAL TEST MEALS WITH SOME ANIMAL EXPERIMENTS.[1]

By JOHN BOSTOCK, M.B., B.S. (London), D.P.M.,
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Introduction.

ALTHOUGH it may seem at first sight a far cry from the psychoses to the digestion, a little consideration will show that the connexion between mind and gastric functions is one of the most intimate.

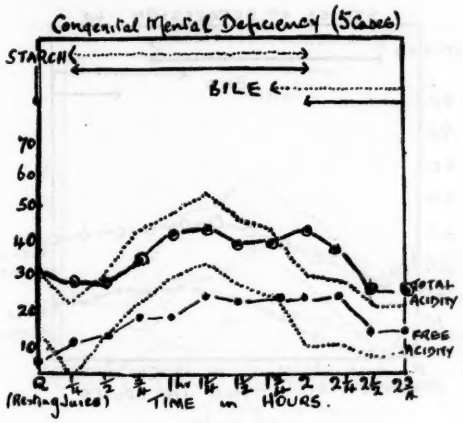
It does not need a psychologist to analyse the reasons for the choosing of "a dinner" to form the background of a convivial evening, for we at once realize that the art of eating pleasantly tinctures our consciousness. The astute man who wishes to impress his client gives him rare meats and then, after the appetite is agreeably satisfied, talks business and every woman knows that she must "feed the man" to have contentment in the home. In truth this business of eating and drinking makes not the least important part of our working life and I am afraid also our "æsthetic" lives! It is only the fact that most of us are comfortably "fed" which disguises their importance. I have recently read an account[1] of the Russian famine in which the psychic effects of hunger have led to the most unexpected behaviour—religious and moral commands and superstitions are annihilated, so much so that women frequently kill and eat their own children! Need more be said?

It is the fashion at present to delve deeply into the instinct of sex. I do not think that it needs an apology if for once we study certain aspects of that other "instinct" which has for its emotional concomitants hunger and repletion.

The literature concerning the gastric functions in mental disease is surprisingly limited (although I confess my inability to cover the ground) and may I be permitted to make a digression in order to emphasize the great need there is for an up-to-date cross indexed medical library. The researcher today loses hours of time and as often as not fails to find the information for which he searches.

The quotations I have found are usually the reflection of current opinion rather than of special investigation. Ford Robertson[2] in 1900 refers to the numerous observations that have now been made upon the composition and toxicity of the gastric juice and emphasizes the latter factor by reference to one Marro[3] who cures by washing out stomachs. Stoddart[4] as late as 1923 in his textbook discusses the digestive processes of the melancholic and manic, infers that there is an increase of hydrochloric acid, a deficiency of pepsin and a greater toxicity. Recently Cotton[5] searching for the toxic factor discovers infection of the stomach in no less than

[1] Read at a meeting of the Sections of Neurology and Psychiatry and Pathology of the New South Wales Branch of the British Medical Association on October 15, 1925.



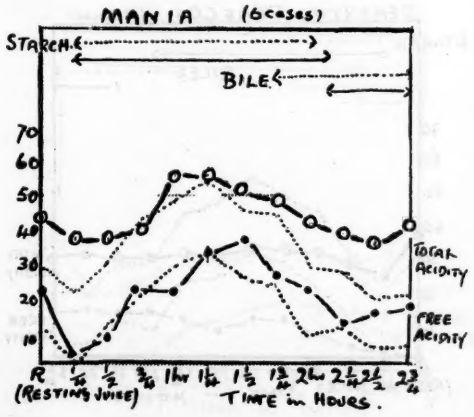
GRAPH I.

83% of male patients and about 76% of females. He states that many manifest a lower free acidity value than is found in controls. The latter of these findings had, I am glad to say, been demonstrated previously in New South Wales by one of our own members, Dr. O. Latham.[6]

The Method.

Using the fractional method of Rehfuss I have endeavoured to investigate the gastric function in the psychoses and this paper describes the results of one hundred consecutive test meals given to eighty-four patients, chiefly in the admission wards of Callan Park Mental Hospital. Nearly all the patients were "certified" and the series represents the graver forms of mental disorder.

When a test meal is to be given, no purgatives are administered overnight and about 9 a.m., whilst fasting, the patient swallows a fine rubber stomach tube and at quarter-hour intervals about five cubic centimetres of stomach contents are abstracted by pumping with a syringe. After the residual gastric juice has been taken away, a feed of thirty-six grammes (one and a quarter ounces) of fine sifted oatmeal boiled in half a litre of water is then given,

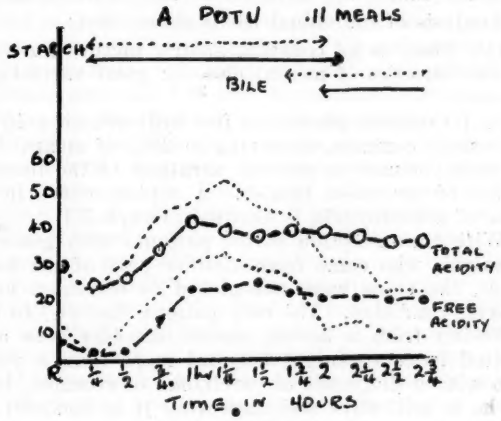


GRAPH II.

the tube being *in situ*. The meal is unsalted. The procedure is simple, but requires no little patience, especially in psychotic subjects. The gastric juice was tested in the usual manner for free acidity by Topfer's reagent and for combined acidity by phenolpthalein. The patients were naturally not selected because of gastric symptoms, but on the score of practicability; often they were too restless, excited or deluded and the test had to be abandoned. That this series was completed is largely due to the good work of my staff who showed unfailing tact and enthusiasm in a very tedious business.

Results.

The results are tabulated in the accompanying graphs. In all the graphs the small dotted line represents the normal reading. The line of dashes and circles represents total acidity and the line of dashes and large black dots represents free acidity. The figures on the abscissa allude to acidity of a one hundred cubic centimetre test meal in terms of decinormal hydrochloric acid. The "norms" are to



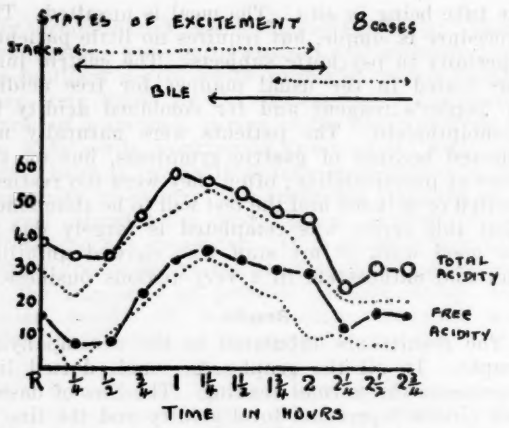
GRAPH III.

be regarded as mere approximations and are inserted as in the diagrams for the sake of convenience for contrast. The great variations make a rigid normal curve non-existent.

An analysis of composite curves shows that:

(i.) In congenital mental deficiency, five meals (Graph I.), mania, six meals (Graph II.), a potu, eleven meals (Graph III.), states of excitement, eight meals (Graph IV.), the curves differ but slightly from what is regarded as normal.

(ii.) Definite lowering of the curves for both free and total acidity are found in the following conditions: Stupor, six meals (Graph V.), states of affective depression, forty-nine meals (Graph VI.), *dementia præcox*, nine meals (Graph VII.), general paralysis of the insane, seventeen meals (Graph VIII.), hypothyreoid, thirty-eighty meals (Graph IX.) and in that of the whole series, one hundred meals (Graph X.). In all of these the rise in acidity is delayed. The curves tend to be flatter than those usually described. Since the onset of



GRAPH IV.

bile is retarded and the acidity at two and three-quarter hours after the ingestion of food is relatively high, it is suggested that emptying of the stomach does not take place until later than in the normal.

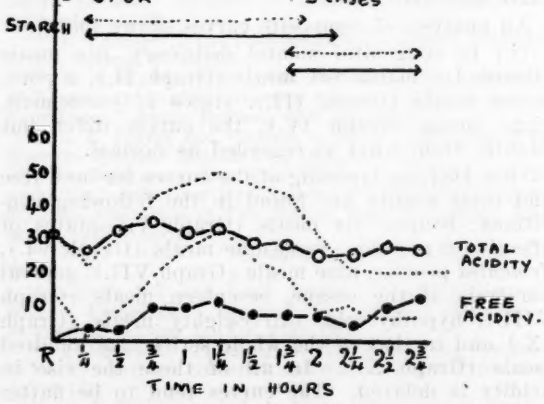
Analysis of individual meals shows that:

(i.) There is no constant gastric picture for any of the varieties of mental disease; great variations occur.

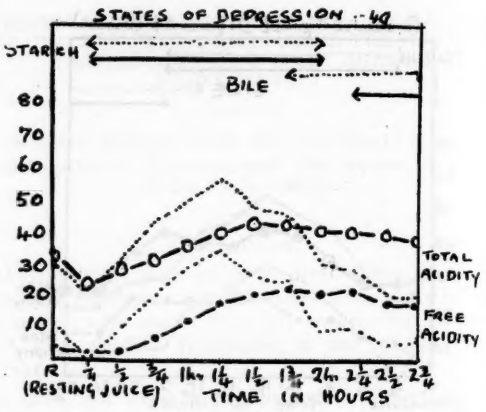
(ii.) Complete absence of free hydrochloric acid is extremely common, occurring in 25% of meals. It is most common in general paralysis of the insane (47% of seventeen meals). A typical curve in a case of achlorhydria is shown in Graph XI.

With the exception of the patients with general paralysis who came from another part of the hospital, the cases were not picked to illustrate any special condition. The only patient "known" to be suffering from a severe gastric disorder was admitted from a general hospital more than a year ago with a diagnosis of carcinoma of stomach, but as he is still alive and improving it is doubtful if this was correct.

(iii.) Hypochlorhydria occurred in 34% of the series and was most common in states of affective depression.



GRAPH V.



GRAPH VI.

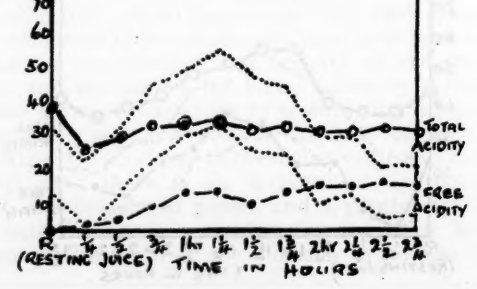
The results of the one hundred test meals are shown in detail in Table I.

Note that in Table I. the cases may appear more than once, for example, a patient may be subject to hypothyreoidism and be in a state of affective depression. Hypochlorhydria is charted when the free acid does not reach the twenty mark on the chart and hyperchlorhydria when it is forty or over. The distinction is chosen arbitrarily for the purpose of this table.

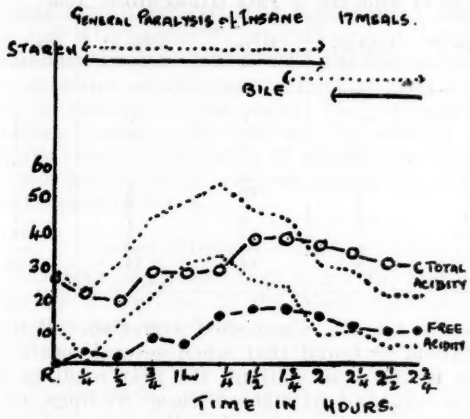
The Interpretation of Results.

The physiology of the gastric juice is a long story which commenced with the ingenious method of research used by Reamur and Spallazani and was continued by such eminent investigators as Alexis St. Martin, Pavlov, Heidenhain to mention but three. Time will permit of only a cursory glance, but an excellent summary by A. J. Carlson[7] of the state of our knowledge up to a couple of years ago will be found in *Physiological Reviews*.

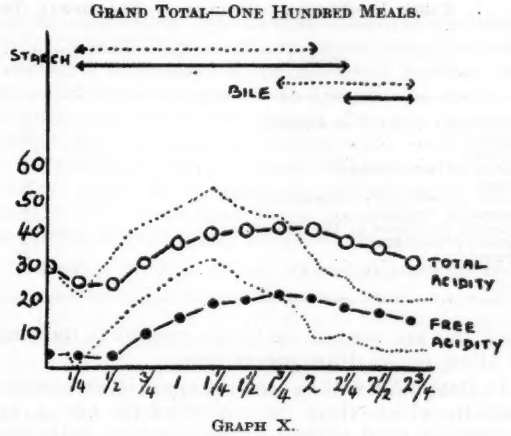
The salient features are the differentiation by Pavlov of an appetite juice secreted on the sight or smell of food through the action of the vagus nerve and a further secretion which is carried on whilst the food was in the stomach. In such a work



GRAPH VII.



GRAPH VIII.



GRAPH X.

as that of Starling's "Physiology of Digestion"[8] this latter flow is accepted as being due to the activity of a hormone (gastrin) discovered by Edkins. The products of digestion send a "chemical messenger" into the blood which provokes further secretions. Unfortunately, adverse criticism has since been levelled against this charmingly simple conception and the position is not now so clear. It is stated by Lim[9] and others that a form of nervous mechanism is involved; moreover, gastrin is not specific and it has been shown that histamine has similar properties.

At this stage it will be useful to state what are the main factors operative in the secretion of gastric juice.

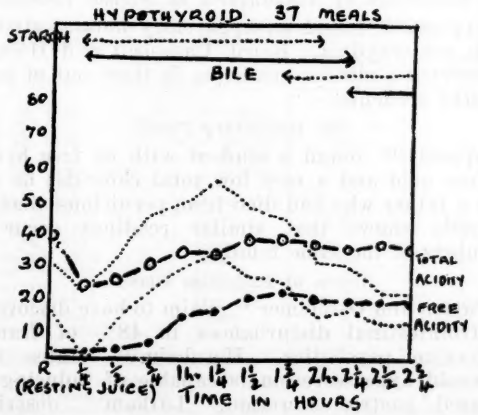
Mode of Secretion of Gastric Juice.

The mode of secretion of the gastric juice is not fully known.

Motility of the Stomach, the Opening of the Sphincters and the Alkaline Duodenal Reflux.

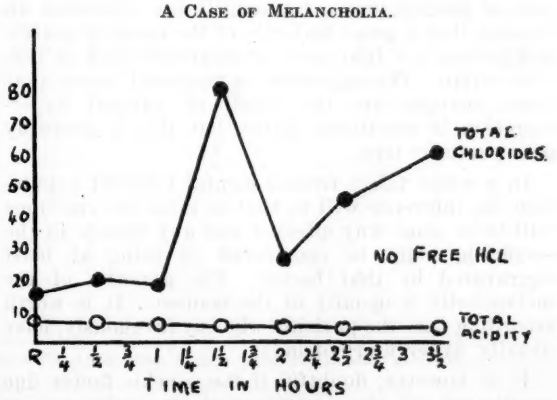
It will, I think, be obvious that if the stomach empties quickly its contents will be altered. Bolton[10] in England and Apperly[11] in Australia have done most important work in showing that the gastric acidity is changed by the reflux from the duodenum and that this regulates the opening of the pyloric orifice. To demonstrate this point it is necessary to estimate the total chlorides and they find that an apparent absence of free hydrochloric acid may be illusory in that it has been present, but has rapidly neutralized. Unfortunately, the older methods of total chloride estimation are cumbersome and I greatly regret that this investigation was not carried out in my series of cases. However, the fact that the bile flow is delayed in the majority suggests that the duodenal reflux is not entirely to blame for the achlorhydria. Admitting the importance of total chloride estimations it must not be forgotten that, as shown by Knott,[12] hydrochloric acid is an excellent antiseptic. Coombe[13] and more recently Hurst[14] have called attention to the need for combating this lack as one likely to produce intestinal intoxication by the overgrowth of the bacterial fauna. I may say that since working on this series I prescribe dilute hydrochloric acid in doses up to six cubic centimetres (one and a half fluid drachms) much more freely than hitherto.

Recently Dr. Latham has performed for me some total chloride estimations and the results are high, although the free acidity is low. They certainly suggest that the reflux factor is of great import-



GRAPH IX.

A CASE OF MELANCHOLIA.



GRAPH XI.

TABLE I.—SHOWING RESULTS OF FRACTIONAL TEST MEALS AS TO AMOUNTS OF FREE HYDROCHLORIC ACID.

Disease.	Number of Meals.	Number of Cases.	Achlorhydria.	Hypochlorhydria.	Normal Limits.	Hyperchlorhydria.
Congenital mental deficiency	5	5	—	1	2	2
Mania	6	6	2	—	—	4
A potu	11	11	1	5	2	3
States of excitement	8	8	2	—	—	6
Stupor	6	5	2	3	—	1
States of affective depression	49	39	9	19	6	15
Dementia Præcox	9	8	2	4	2	1
General paralysis of the insane	17	12	3	4	1	4
Hypothyreoidism	37	29	6	15	5	10
Myxœdema	2	2	2	—	—	—
Total of meals in series	100	84	25	34	14	27

ance, but my number up to the present is too small to allow me to draw conclusions.

In Graph XI will be seen the chart of an excellent case in which there is achlorhydria, but a fair amount of total chlorides, the inference being that the achlorhydria is not an expression of absent secretion.

Case A.Y., a female, married, aged fifty-seven years, was admitted on April 29, 1925, with acute melancholia, delusions of unworthiness and a history of attempted suicide. She complained of gastric symptoms, slight indigestion which was considered to be part of her psychosis. It may be noted that exhibition of dilute hydrochloric acid in six cubic centimetre doses did not materially relieve her condition and there is profound anorexia; the patient states she has no desire for food of any kind.

The Action of the Vagus Nerve.

According to Langdon Browne[15] the vagus is motor to the secreting glands, œsophagus and pyloric end of stomach. If the vagi are severed, no appetite secretion of gastric juice occurs.

The Action of the Sympathetic Nerves.

The impulses of the sympathetic nerves are said to inhibit secretion and movement of pyloric half of stomach.

The Psyche.

It has long been known that in both animals and man the gastric peristaltic waves and secretion of gastric juice are affected to a marked degree by emotion. A research by Heyer[16] has demonstrated a law that any diversion of the mind, painful or pleasurable, from the act of eating checks the secretion of gastric juice. Cannon[17] has expressed an opinion that a great majority of the cases of gastric indigestion are functional in character and of nervous origin. The suggestion is frequently made that these changes are the result of adrenal hypersecretion in emotional states, but this is probably only partially true.

In a series taken from a mental hospital population the inference will be that in most the emotions will be in some way affected and any change in the secretion might be considered as being at least aggravated by that factor. The appetite of the melancholic is usually of the poorest. It is worth recording that the patients who eat ravenously, have usually hyperchlorhydria!

It is, however, doubtful if the psychic factor due to disgust at the insertion of the tube *per se* accounts for the diminished secretion. Bell and MacAdam[18] found that when several meals were given to the same patient, the first readings were the lowest and attributed these findings to the immediate psychic influence. My own observations show that the first meals tend to have the highest curves which would, if anything, discount this immediate emotional factor.

Many psychotic subjects are indifferent to their surroundings and the presence or absence of food causes no "appetite" response. This is notably the case in stupor and states of depression. I am inclined to think that the emotional factor, dependent upon hunger, is of far greater importance in test meal observations than has hitherto been suggested.

The Action of "Gastrin."

As already stated the action of "gastrin" is *sub judice*.

Normal Variations in Acidity.

Bell and MacAdam have pointed out how very variable are the findings in normal people, but that none the less the curves usually tend to approximate. I have confirmed these observations, several of my patients manifesting considerable differences. Although these facts make the single test meal of less value, the manner in which the one hundred analyses here depicted fall into a definite curve, is evidence of the existence of a definite tendency and show that the results may yet have a scientific worth.

The Occurrence of Achlorhydria in Normal Individuals.

Apperly[19] found an apparently normal student with achlorhydria. Baird, Campbell and Hern[20] discovered a similar condition in three out of sixty healthy students.

The Hereditary Factor.

Apperly[19] found a student with no free hydrochloric acid and a very low total chloride; he had lost a father who had died from pernicious anæmia. Apperly shows that similar readings occur in members of the same family.

Action of Endocrine Glands.

Patten and Ostheimer[21] claim to have discovered gastrointestinal disturbances in 48% of frankly endocrine psychotics. Hernheim[22] states that thyreoid hypersecretion is capable of inducing increased gastric secretion. Latham[6] described achlorhydria in three cretins and I have found it

similarly in two patients with myxœdema. Rogers, Rabe and Ablahadian[23] injected organic extracts subcutaneously and found that the thyreoid can cause secretion of the gastric juice in part by its action on filaments of the vagus. They showed that inhibition occurs with extracts of adrenal and pituitary tissue. The action of adrenal has already been mentioned and it suggests again the field of vago-sympathetic-tonus.

Effect of General Nutrition.

It has been suggested that a general metabolic change or alterations in the blood content may influence gastric secretion.

Experimental Error.

The experimental error includes the ingestion of saliva which may vitiate results. Kopeloff[24] claims that this factor is responsible for the bacterial content of the gastric juice. The methods of tituration and choice of indicators have stood the test of time and are reliable for practical purposes.

I will candidly admit that when I perhaps too light heartedly commenced this investigation, it was with an inadequate knowledge of the difficulties of interpretation of whatever results I might obtain. It is sometimes difficult to assess two accessory causal factors, but their multiplication makes a puzzle suitable to the taste of a cross-word enthusiast. The formidability of the list is depressing, for it means that an exhaustive research on the gastric functions of the psychoses must be a lengthy business, requiring the help of the biochemist, the physiologist, radiologist and the like. Such assistance being unfortunately not at present available, I have drawn a bow at a venture in an attempt to elucidate at least one aspect of the problem.

Is There a Relationship Between the Thyreoid Gland, Vagal Nerve Nucleus and Digestion?

To my mind one of the most remarkable facts in psychiatry is the extraordinary tolerance to thyreoid feeding displayed by mental hospital patients. Clinically mild degrees of hypothyreoidism are very prevalent. Kojima[25] by pathological findings has borne out this contention. In the present series of patients who were not chosen for any thyreoid changes, no less than twenty-nine were noted by me to be clinically hypothyreoid subjects either by physical signs or tolerance to the extract. Is it possible that there may be a connexion between the gastric secretion and the products of this gland?

J. Walker[26] has shown that 50% of patients with *dementia præcox* have a low basal metabolic rate, but he will not admit this to be due to hypothyreoidism, because thyreoid feeding does not cure it. He, therefore, postulates that the trouble is due to an underlying nervous cause! In other words, he regards the thyreoid not as a universal catalyst or energiser, but as acting through an intermediary.

Similarly one may at once say that the thyreoid is not directly concerned with digestion. Exhibition of the extract by the mouth to man or rabbit I found to have no influence on the amount of free hydrochloric acid.

As the appetite juice undoubtedly arises through the intermediary of the vagus, this great nerve is the first obvious link to be investigated.

It is permissible at this stage to make two tentative speculations:

(i.) If the vagal nucleus were not functioning correctly, then less free hydrochloric acid would be secreted and there might be in addition alteration in gastric and pyloric motility which would lead to differences from the normal in the composition of the gastric juice.

(ii.) There may be a relationship between the thyreoid gland and the *nucleus dorsalis vagi* or a basal nucleus subserving the function of the "appetite" emotion.

It must be clearly understood that I do not refer to the vago-sympathetic-tonus conceptions of Eppinger and Hess which in spite of their popu-

TABLE II.—SHOWING RESULTS OF FRACTIONAL TEST MEALS ON RABBITS WHOSE THYREOIDS HAD BEEN REMOVED, WITH REFERENCE TO FREE HYDROCHLORIC ACID AFTER A LUCERNE MEAL.

Date of Examination.	Rabbit.	Time in Hours.						
		Quarter Hour.	Half Hour.	One Hour.	One and a Half Hours.	Two Hours.	Three Hours.	Five Hours.
April 6, 1925	F. White			0		0	0	+
April 9, 1925	F. White				0		0	0
April 16, 1925	F. White	0	0	0				
April 20, 1925	F. White	+		+				
April 22, 1925	F. White			0				
April 6, 1925	Blue			+		+	0	+
March 26, 1925	Blue				0		0	+
August 10, 1925	Blue	0	0	+				
August 14, 1925	Blue	0			+		+	
March 26, 1925	Red				+		0	0
August 10, 1925	Red	+	0	+				
August 14, 1925	Red	+			0			
August 10, 1925	Brown	+	0	+				
August 14, 1925	Brown	0	0	+			+	
August 10, 1925	Black	0	+	0				
August 14, 1925	Black	+		+			0	

Dates of dethyreoidectomy: White, February 26, 1925; Blue, March 19, 1925; Red, March 12, 1925; Brown and Black, April 21 and 23, 1925.
The cross denotes presence of free hydrochloric acid, absence by 0.
A small series of control tests on normal rabbits gave 72% with free hydrochloric acid present as against 46% in the animals whose thyreoids had been removed.

larity are open to serious objections. I regard the thyreoid as neither stimulator nor depressor, but as a something necessary towards correct vagal functioning. It is, moreover, highly improbable that the thyreoid and the vagal nucleus are the only two factors concerned.

There is already evidence from pathology that some such relationship is present, since Mott[27] has described three cases of spontaneous hypothyreoidism in which were found universal chromatolytic changes in cells of the central nervous system, especially smaller cells of the autonomic nuclei, for example, vagus and glossopharyngeal.

I have had no opportunity of carrying out similar observations, but during the early part of this year a chance presented itself whereby I could attack the problem from the biological point of view, as I was able to perform thyreoidectomy on five rabbits at the laboratory of the mental hospitals. Using a gag, a small rubber catheter and a "record" syringe, it was not difficult to abstract a little fluid from the stomach of these animals, so by this means I hoped to gain first hand information as to any relationship between thyreoid activity and gastric digestion. Moreover, by cross sectioning the brain, it might be possible to detect changes in the vagal nuclei.

The results of the investigation into the gastric acidity are tabulated in Table II. The first test was dramatic, giving achlorhydria in the animal whose thyreoid had been removed, whilst the control yielded free hydrochloric acid at every stage of the meal. Any jubilation upon my part was, however, premature since subsequent meals yielded considerable variation in the results. The most that can be inferred from this small series is that achlorhydria is more common in those animals which have lost their thyreoid gland, but this is by no means constant. It is worth recording that in one animal it occurred in no less than ten out of thirteen tests and that this individual most nearly approximated to a myxœdematous condition, being dull and lethargic. The other rabbits showed little variation from their usual liveliness and it might be asked if they were really hypothyreoid and whether operative technique was not faulty?

Vincent and Jolly,[28] however, have observed that monkeys, rats, guinea pigs are often not inconvenienced by removal of the thyreoid gland and came to the conclusion that this gland is not essential to life, the pathology of myxœdema must be more complex than simple insufficiency of thyreoid material. Carlson and Woelful[29] are said to have reported that myxœdema does not develope in rabbits whose thyreoids have been removed, at least in seven months.

Such are the difficulties which beset the researcher in endocrinology! The second phase of the investigation which concerns the examination of cross sections of the base of the brain, has not yet been finished. Finally, I may add that I am acutely aware of the limitations of this presentation. However, I have consoled myself with the thought that half a loaf is better than no bread! Possibly it is justified, if it only serves as a reminder of the many complex factors which are found in even the simplest problems of our speciality.

Conclusion.

1. Achlorhydria and hypochlorhydria are discovered to be surprisingly frequent in the psychoses; it is not inferred that they are causal, but that they are expressions of the general psycho-physical change.

2. The mode of production of these physiological changes is not precisely determined, but it is probable that a large number of factors are involved, the most important of these being:

   (*a*) The psyche including alterations in the emotion of hunger.

   (*b*) Changes in the motility of the stomach, the sphincteric mechanisms and the duodenal alkaline reflux.

3. Evidence is adduced concerning the possibility of an intimate functional connexion between the vagal nucleus, the thyreoid gland and digestion. A preliminary investigation shows that achlorhydria is relatively more common in rabbits whose thyreoids have been removed than in normal controls.

Acknowledgements.

I desire to acknowledge my indebtedness during the research to Professors H. Priestly and D. A. Welsh and Dr. S. Dodd, of the University of Sydney, for valued advice; to Dr. S. E. Jones for placing considerable information at my disposal; to Dr. O. Latham for his help on many problems and to his assistant, Mr. F. Hedger, for his aid in the animal experiments and to the staff of the Fisher Library for their courtesy and assistance. My debt to the staff of Callan Park Hospital has already been mentioned. Finally, I must add my thanks to Dr. J. A. L. Wallace, Medical Superintendent of Callan Park Hospital, for his practical encouragement and permission to publish.

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A SIMPLE TECHNIQUE FOR THE ESTIMATION OF THE BLOOD UREA: THE HENCH-ALDRICH METHOD.

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HENCH and ALDRICH have recently applied the principles employed in their method of estimating the salivary urea index,[1] the value of which has been confirmed by Corkill[2] and other observers, to the direct estimation of the urea content of the blood.[3] Only the simplest apparatus is required for the test, an ordinary burette, a five cubic centimetre pipette, a funnel and filter papers. The reagents are 10% dilution of trichloracetic acid, 5·0% solution of mercuric chloride which needs to be very accurately made up and saturated solutions of sodium carbonate and potassium oxalate. The only disadvantage of the method is that a relatively large sample of blood has to be obtained, eight cubic centimetres for the filtration method or five cubic centimetres if a centrifuge be available.

In the present paper we compare the results of this test with those obtained by Maclean's modification[4] of Van Slyke's method.[5] One of us (K.D.F.) was responsible for the Hench-Aldrich, the other (B.S.) for the Maclean method. The former test, except in three cases (60, 61 and 62 in Table III.) was carried out without knowledge of the values yielded by the other.

Technique.

A drop of 20% potassium oxalate solution is placed in the tube in which the blood is to be collected, and immediately after the blood has been added the tube is rapidly inverted a few times to prevent clotting. To eight or ten cubic centimetres of the oxalated blood in a test tube or to five cubic centimetres in a centrifuge tube is added an equal quantity of 10% trichloracetic acid and the contents are mixed by inverting the tube a few times. A dirty brown precipitate is formed. If the larger amount of blood is available, the mixture is filtered; otherwise it is centrifuged for five to ten minutes, the supernatant fluid is poured off and either filtered or cleared by centrifugation. It is essential to obtain a filtrate absolutely free of any trace of protein and preferably five cubic centimetres in amount. It is advisable to use a small filter paper so that there will be as little absorption of filtrate by the paper as possible. Five cubic centimetres of this filtrate are placed in a small evaporating dish and from a burette two cubic centimetres of a 5% solution of mercuric chloride are added, the mixture being stirred while the mercuric chloride is being added. A drop of the mixture is then taken and added to a drop of the saturated solution of sodium carbonate on a white porcelain tile. The end point is indicated by the appearance of the first faint tinge of brown appearing within three seconds. If the end point is not reached, 0·1 cubic centimetre of the solution is added from the burette and a second drop of the mixture is tested. This process is repeated until 2·6 cubic centimetres have been run in. Thereafter 0·2 cubic centimetre of the mercury solution is added at a time until the end point is reached. With five cubic centimetres of filtrate, the figures for the normal blood urea (twenty to forty milligrammes per hundred cubic centimetres) fall between 2·0 and 2·5 cubic centimetres of the 5% mercuric chloride solution. With increasing experience it is possible to detect a high reading on adding the first drop of the mixture to the carbonate. A white precipitate appears and with further additions of mercuric chloride the colour

TABLE I.

Number.	Milligrammes of Urea per 100 Cubic Centimetres of Blood.		Difference in Milligrammes.
	Maclean's Method.	Hench-Aldrich's Method.	
1	38	40	+ 2
2	30	36	+ 6
3	15	24	+ 9
4	27	32	+ 5
5	13	19 [1]	?
6	20	20	0
7	18	26	+ 8
8	38	48	+ 10
9	23	20	− 3
10	23	28	+ 5
11	24	22	− 2
12	25	28	+ 3
13	29	27	− 2
14	28	29	+ 1
15	21	24	+ 3
16	39	39	0
17	39	44	+ 5
18	36	40	+ 4
19	13	15	+ 2
20	18	23	+ 5
21	35	33	− 2
22	29	27	− 2
23	18	20 [1]	?
24	36	39	+ 3
25	32	37	+ 5
26	19	20	+ 1
27	32	32	0
28	17	17	0
29	29	33	+ 4
30	24	23	− 1
31	38	40	+ 2
32	34	28	− 6
33	23	25	+ 2
34	24	26	+ 2
35	34	36	+ 2
36	31	33	+ 2

[1] 19— 20— mean that the first drop tested showed the end point had been passed and the value was less than that recorded.

changes through pale and gradually deeper yellow to the end point. In such a case the mercuric chloride solution is at first added in amounts greater than 0·1 cubic centimetre.

When the blood urea values are very high (over 150 milligrammes per hundred cubic centimetres) this method gives results some fifteen milligrammes or more below Maclean's method. This is due to the relatively large number of drops used for testing. Much closer agreement could be obtained if a ranging sample were tested before the final estimation was made. The error in these high readings is, however, of litttle clinical importance.

Unless the blood is collected under sterile conditions and kept in an ice chest, it is advisable to estimate the blood urea within a few hours of obtaining the sample.

Calculation.

As the blood was originally diluted by an equal volume of acid, the five cubic centimetres filtrate corresponds to 2·5 cubic centimetres of blood; therefore to obtain the mercury combining power of one hundred cubic centimetres of deproteinized blood, the titration value must be multiplied by forty. Now the mercury combining power of the "rest nitrogen" of this volume of blood is equal to sixty cubic centimetres of 5% mercuric chloride solution, a value which remains practically constant under all circumstances (Hench and Aldrich private communication).

If two cubic centimetres of a 5% mercuric chloride solution are used for five cubic centimetres of filtrate, one hundred cubic centimetres of the deproteinized blood in question would require eighty cubic centimetres of the reagent. Subtracting from this the mercury combining power of the "rest nitrogen" we obtain a value which practically coincides with the blood urea, since one cubic centimetre of 5% mercuric chloride solution is equivalent to one milligramme of urea. The blood urea in this case would be approximately twenty milligrammes per hundred cubic centimetres. Briefly, if x cubic centimetres of a 5% mercuric chloride solution are used for five cubic centimetres of filtrate, the blood urea determined in this way would be $40x$—60 milligrammes per hundred cubic centimetres.

The results recorded in milligrammes of urea per cubic centimetres of blood have been presented in the following tables. In Table I. are given the values which fall within normal limits arranged in the order in which the estimations were made. It is seen that with increasing experience of the test the results become more accurate.

In Table II. are grouped the figures from samples of blood with moderately raised urea content (forty to one hundred milligrammes per hundred cubic centimetres) in an ascending scale of blood urea values. The greatest difference in results in this series was obtained in the first one tested (number 45).

In Table III. are the samples with very high urea values, exceeding one hundred milligrammes per hundred cubic centimetres of blood. Number 58 was the first one tested in this series. In numbers 60, 61 and 62 the results of the Maclean determinations were known and no test drops were taken until the dilutions were somewhere in the vicinity of the expected end point. These results show that the low readings ordinarily seen with the Hench-Aldrich method in samples of blood with high urea values are due to the numerous drops removed in testing for the end point, hence the advisability in such circumstances when greater accuracy is desired, of testing a ranging sample first. For clinical purposes, however, this is unnecessary.

Our results clearly demonstrate that for all clinical purposes the values obtained by the Hench-Aldrich method are comparable with those deter-

TABLE II.

Number.	Milligrammes of Urea per 100 Cubic Centimetres of Blood.		Difference in Milligrammes.
	Maclean's Method.	Hench-Aldrich's Method.	
37	41	45	+ 4
38	41	40	− 1
39	42	46	+ 4
40	44	36	− 8
41	46	46	0
42	47	45	− 2
43	60	70	+ 10
44	63	60	− 3
45	68	56	− 12
46	71	64	− 7
47	76	70	− 6
48	79	75	− 4
49	83	77	− 6
50	91	100	+ 9

TABLE III.

Number.	Milligrammes of Urea per 100 Cubic Centimetres of Blood.		Difference in Milligrammes.
	Maclean's Method.	Hench-Aldrich's Method.	
51	110	124	+ 14
52	118	112	− 6
53	165	160	− 5
54	182	169	− 13
55	194	180	− 14
56	230	208	− 22
57	256	220	− 36
58	434	372	− 62
59	450	430	− 20
60	105	105	0
61	241	238	− 3
62	252	248	− 4

mined by Maclean's method. Where the advisability of a blood urea determination is doubtful, we should advise the estimation of the probable blood urea by the Hench-Aldrich salivary urea index method.[1] This gives sufficient information in all cases in which the salivary urea reading is well above or far below the upper limits of normal. But where the values are in the vicinity of the upper limits of normal (a little above or below) it is always advisable to confirm the results by a direct estimation of the blood urea content by the simple method outlined above. In comatose patients, the direct blood urea test would obviously be necessary.

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Our thanks are due to the members of the honorary staff of this Hospital and to the Medical Superintendent for making the material available and to the Director of the Walter and Eliza Hall Institute for facilities he has placed at our disposal.

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NOTE ON THE PRESENCE OF "CERCARIA PIGMENTOSA" IN LIMNÆA BRAZIERI BRED IN AN AQUARIUM TO WHICH SHEEP FLUKE EGGS HAD BEEN ADDED.

By BURTON BRADLEY, M.B. (Sydney) D.P.H. (London),
Sydney.

IN a previous communication I gave the results of an epidemiological and biological investigation into the relationship of various snails to the sheep fluke trouble and as a result I claimed to have made out a strong *a priori* case that a particular snail, *Limnæa brazieri* was the chief transmitting intermediate host of *Fascioli hepatica* in this State. Subsequently McKay claimed "conclusive proof" that *Limnæa brazieri* was a transmitting agent. I have already shown that his published facts warranted no such sweeping statement. The present note does not claim that the evidence to be disclosed constitutes "conclusive proof," but it, at any rate, tends to confirm my previous work that a particular cercaria named by myself provisionally *"Cercaria pigmentosa"* is probably the same as that described by Thomas and is probably the larval stage of the sheep fluke thus tending also to confirm the general hypothesis as to the relationship to fluke of *Limnæa brazieri.*

The new evidence is as follows:

On December 5, 1925, a few snails (about three dozen in all) were recovered from a bog which had become much overgrown with weed and grass. On previous occasions it had usually been easy to collect numerous snails from this spot. *Limnæa brazieri* alone were found on each of five occasions in 1925 (on June 9, September 9, September 29, November 11 and December 5). The cercaria was found in June (one snail infected among sixty examined) and in November (four snails infected among one hundred and twenty-five examined). Nineteen of the snails collected on this last examination of the spring made on December 5, were individually examined and no *Cercariæ pigmentosæ* or other cercariæ were found. At least half of these snails were small, being about three or four millimetres long in the spiral axis.

The remainder were mostly very small, some being only about two millimetres and were diffult to count and handle. These were put into an aquarium which had been established about two weeks. This apparatus consisted of a glass cylinder into the bottom of which about an inch of black soil from the laboratory garden had been placed. It was filled with water to a depth of seven to twelve centimetres and in the soil was planted some water weed the fronds of which eventually extended to the surface of the water projecting slightly. The water weed was obtained from a weedy creek in which there were habitually *Limnæa brazieri*, but was well washed several times and no snails were noted on it by naked eye examination. These facts have an obvious importance.

The exact number of snails added to the jar was not counted; the presence of soil and the smallness of the individual snails making this difficult. There would be rather less than twenty snails, unless there were almost microscopic specimens present.

Several of these snails probably died, but on January 21, 1926, there were found to be present several snails about seven millimetres long. The growth of water weed made accurate counts impossible without disturbing the aquarium. The snails periodically descended into the depth, lying on the soft black mud and being difficult to see. There were at least seven medium sized snails noted. On this date, January 21, 1926, fluke eggs were obtained from a sheep's liver and after washing these in

water, a thickly opalescent emulsion was made. Of this about two cubic centimetres were added to the aquarium which now contained about a litre of mud and water. Towards the middle of February about twenty very small snails were seen in the jar. These were about two millimetres long. In view of the rapid development of the first lot of snails put in and the time elapsing, these snails would seem either to be the progeny of the first snails or to have developed from eggs accidentally included. There were in fact probably one or two egg masses poured in with the first snails.

On March 31, 1926, examination was made of two large snails and two of the smaller snails. In the latter "pigmented" cercariæ of the type described as *"pigmentosa"* were found. On April 3, 1926, the majority of the small snails recoverable from the jar were individually examined in the usual way for cercariæ.

The results were as follows:

Number of snails examined 32.

Number infected with *Cercaria pigmentosa* 13 (40%). Of these eight were in an immature stage, but definitely of the same type.

Rediæ were noted of usual type in many cases. In one snail cysts of a dark colour were seen, but no cercariæ. No other types of cercariæ were found.

Examining the salient facts of the above described events there seems no reasonable doubt that the cercariæ discovered in the smaller snails ten weeks after they had been mixed with the fluke eggs were derived from those eggs, that is, were the cercariæ of the sheep fluke, *Fasciola hepatica*. It is, of course, obvious that there are one or two possible fallacies, but they are more fanciful than real. One would have to explain the very large infestation rate (13 in 32 or about 40%, an infestation rate never discovered for this species in any natural water collection during the examination of more than four thousand *Limnæa brazieri*) by supposing that the small snails containing the cercariæ were snails put in originally in a very young state, already infected with at least ten times as high a rate than their fellows in the pond. This is a possibility indeed which might have been brought about by some special circumstance and which is admittedly not completely controlled out by the negative findings in the nineteen controls. These were generally larger snails and in any case very early stages of fluke infestation in snails is readily missed. It requires, however, considerable faith in the long arm of coincidence to believe that in just this circumstance snails naturally infected should show more than double the highest rate of infestation ever found by me for this species of cercaria and more than twenty times the usual average find. The very high rate is just, however, what one would expect under the described experimental conditions. It would be very difficult to conceive that the snails that are now being examined, were some of the originals added as it would involve a very slow growth rate and side by side in the aquarium were the larger snails that have grown at apparently normal rates.

Unless egg infection is possible, this explanation seems almost inconceivable. Other possible fallacies such as the accidental introduction of eggs in dust from other trematodes seem chimerical. As I have in my previous communication to some extent described the cercaria with which we are here concerned and am shortly publishing fuller descriptions and photographs, there is really no need to do so here as the cercaria has been compared by me with type specimens of Cawston and appears in every way similar. The descriptions by Faust should be consulted. The cercaria agrees with the original description of Thomas and all measurements I have made coincide.

Conclusion.

Although no "conclusive proof" in the scientific sense can be drawn from this experiment, the most probable explanation of a 40% infestation of *Cercaria pigmentosa* in the aquarium (?bred) snails seems to be that they developed from the miricidia from the eggs put in on January 21, 1926, ten weeks before.

If this is so, it tends to add evidence to the theory first put forward by me and supported so far entirely on the general epidemiological evidence I brought forward that *Limnæa brazieri* is one and probably the chief transmitting agent of sheep fluke in New South Wales.

Reports of Cases.

EPISCLERITIS AND TUBERCULIN TREATMENT.

By J. FRANK SPRING, M.D. (Melbourne),
Honorary Ophthalmic Surgeon, Saint Vincent's Hospital, Melbourne.

MISS X., an apparently healthy young woman, consulted me in January, 1925, with episcleritis. In spite of all treatment and a consultation with a distinguished colleague, her condition was no better six months later.

I had her examined by her own capable physician, who reported: "Nothing abnormal found; no evidence of any tubercular lesion."

In September she had been away for a change and had to return prematurely because her eye was troublesome. Feeling almost at the end of my resources, I decided to try the effect of a course of tuberculin.

Having long held the opinion that tuberculin should only be used in small doses, I decided to start with 0·00001 milligramme T.R. and to give the injection weekly.

The first injection was given on September 8, 1925, the patient being just as bad as she was in January; one week later there was a definite improvement in the eye. The second injection of the same size was also followed by some improvement, but after a third similar injection the eye flared up and was just as bad as it was when she commenced treatment.

I was in doubt as to how to proceed. It seemed certain that the injections had effected some change in the condition, as the improvement in the first two weeks was striking. Even the setback after third injection suggested that this kind of treatment had possibilities. I had now either to increase or decrease the dose. Each line of action had something to be said in its favour, but safety seemed to be in trying a smaller dose first.

Continuing the weekly injections I gave 0·000005 milligramme T.R. next time. This proved satisfactory, but

after three injections the eye, though improved, seemed to come to a standstill. I returned to the injection of 0·00001 milligramme, only to get a rather more vigorous reaction than I desired. Being now satisfied that 0·00001 milligramme was too big a dose, I still thought 0·000005 milligramme too small. Next week I gave 0·000008 milligramme. This gave the very slightest reaction which had subsided in three days, leaving the eye almost well. Three more injections of 0·000008 milligramme completed the cure. On December 8, 1925, the eye looked well and treatment was stopped. It has remained well to date, March 15, 1926, without any further treatment.

This case to my mind is particularly instructive. Not merely because an intractable episcleritis was cured in three months by tuberculin, but rather because of the remarkable difference in the results obtained when minute alterations were made in the size of the dose.

I am not in a position to be positive that the tuberculin injections did not act merely as a foreign protein, but the definite variation caused by the minute alterations in the dose seem to indicate that one had to deal with a specialized action.

I do not see many cases of intractable episcleritis and consequently feel it would be almost impossible for me to prepare a series of cases, so I have not waited to try and do so.

I recognize that one case proves nothing, but I feel that this one suggests so much that it should be published.

Finally I wish to lay stress on the fact that most minute doses were used.

BANTI'S DISEASE.[1]

By R. J. MILLARD, C.M.G., M.B., Ch.M. (Sydney),
Medical Superintendent, The Coast Hospital, New South Wales.

A MALE patient, aged thirty-four years, a secretary, was admitted to the Coast Hospital on August 11, 1925. At the time of admission he said that he had been ailing since early in 1923. Since that time he had lost at least nineteen kilograms (three stone) in weight. He suffered from hæmatemesis in April and September, 1924 and in June, 1925. He had been subject to dizziness and faintness and to chronic constipation. He had been under treatment in the Royal Prince Alfred and in the Sydney Hospitals. He was discharged from the latter institution three weeks before his admission to the Coast Hospital.

On admission he was pale, but not very thin. The liver dulness was reduced and the spleen was very much enlarged. Temperature, pulse and respiration were normal.

On August 12, 1925, his blood was examined. The red cells numbered 4,500,000 and the white cells 2,500 per cubic millimetre. The hæmoglobin value was 80% and the colour index 0·8. Some punctate basophilia with poikilocytosis was present. No nucleated red cells were seen.

On August 13, 1925, the patient was submitted to an opaque meal examination. The stomach was found to be displaced to the right, the greater curvature being pushed in by the enlarged spleen which extended almost to the iliac crest. The duodenum was regular.

On August 19, 1925, there were 5,000,000 red cells and 2,800 white cells per cubic millimetre. Of the white cells 82% were neutrophile cells and 18% lymphocytes. The hæmoglobin value was 85% and the colour index 0·8. A moderate amount of punctate basophilia was present, but very little change in the size or shape of red cells was found. No nucleated red cells were seen.

On August 26, 1925, splenectomy was performed by Sir Alexander MacCormick. The spleen was adherent to the posterior abdominal wall. No splenic hilus was found. The vessels entered the organ over the whole of the posterior surface. These were tied and cut, the spleen was removed and the wound was closed in layers. No drainage was used. For four days after operation there was abdominal distension and much discomfort. The distension was relieved when the bowels acted after administration of a high enema combined with an injection of one cubic centimetre of "Pituitrin" and 0·0006 gramme of eserine.

A report from the Bureau of Microbiology described the spleen as being the seat of generalized fibrosis and sclerosis of its vessels with atrophy of the Malpighian corpuscles, changes considered to be in keeping with a diagnosis of Banti's disease.

On September 8, 1925, a blood examination was made. There were 4,100,000 red cells and 6,800 white cells per cubic millimetre. The hæmoglobin value was 80% and the colour index 0·9. Some irregularity in the shape and size of the red cells was found, but no nucleated forms were seen.

About a week after the operation the patient began to suffer from irregular pyrexia. On September 12, 1925, he had acute pain in the right side of the chest. The sputum was bloodstained. It was examined microscopically on eight separate days between September 14 and October 5, 1925, but no tubercle bacilli were found. By September 22, 1925, the pyrexia had subsided and it did not recur.

On October 7, 1925, the blood was examined. There were 4,960,000 red cells and 8,000 white cells per cubic millimetre. Considerable irregularity in the shape of the red cells was found. No nucleated forms were seen. The hæmoglobin value was 80% and the colour index 0·8. On October 24, 1925, the patient was discharged feeling well.

On November 30, 1925, he was readmitted. He said that two weeks after his discharge he had become weak and ill and that for four days previous to readmission he had had much pain in the left side of the abdomen with repeated hæmatemesis and melæna. He was cold and yellow in appearance. His pulse was weak and tenderness was present in the vicinity of the scar of his operation.

On December 1, 1925, hæmatemesis occurred and calcium chloride was given intravenously.

On December, 2, 1925, the blood was examined. The red cells numbered 1,360,000. The hæmoglobin value was 35% and the colour index 1·2.

On December 3, 1925, his temperature at 10 a.m. was 39·4° C. (103° F.) and at 10 p.m. it was 38·9° C. (102° F.).

On December 10, 1925, he felt much better; there had been no more vomiting and the temperature was almost normal.

On December 16, 1925, he was much weaker and on the following day he died.

[1] The patient herein described was shown at a meeting of the New South Wales Branch of the British Medical Association on October 8, 1925.

Reviews.

PHYSICAL AGENTS IN MEDICAL PRACTICE.

"THE time when physiotherapy meant 'baking and massage' is over." It is to demonstrate that this quotation from its pages is correct that "Physiotherapy," by Harry Eaton Stewart, is presented to us as an up-to-date treatise on the use of physical agents in medical practice.[1]

The author points out that modern medical equipment demands a knowledge of the physical agents in use. It is also necessary to know their modes of affecting pathological states and so altering them that their progress is arrested and in many instances a return to normal achieved.

Just as no practitioner would send a patient to a pharmacist for some drugs without specifically stating the exact drugs required and their quantities so should he not merely order electrotherapy or massage or hydrotherapy without stating the exact requirements as to modality to

[1] "Physiotherapy: Theory and Clinical Application," by Harry Eaton Stewart, M.D.; 1925. New York: Paul B. Hoeber, Incorporated. Royal 8vo., pp. 363, with illustrations. Price: $7·50 net.

be used, time of application, amount of current, temperature and so on.

This is important because the same modality may be used by suitably varying it to obtain very different results. Attention is drawn to this fact particularly in regard to massage and hydrotherapy.

One of the main objects of the book is to help the practitioner in gaining the necessary knowledge to fulfil this ideal by choosing and blending suitable agents for a thorough course of physical treatment when it is indicated.

In the first part of the book sections are devoted to the electrical currents, radiant light and heat, actinotherapy, exercise, massage and hydrotherapy.

Intricate details of apparatus are wisely omitted. Concise references are made to the salient points in the physics and physiological actions of the different modalities.

As the hastiness associated with a large clinic often militates against a good result, the author advises individual attention for each patient and stresses the importance of team work among the interested medical men as a means of improving the benefits derived from these forms of treatment. Physiotherapy covers such a wide field that general practitioners, specialists and those dealing with industrial and athletic injuries are urged to avail themselves of the benefits that these agents have proved themselves capable of giving.

The electrical currents dealt with are the galvanic (continuous, interrupted and wave), faradic, sinusoidal and high frequency (d'Arsonval, Oudin, Tesla). The advantages of the more recently introduced sinusoidal current over the older faradic and interrupted galvanic currents are detailed. In dealing with the high frequency currents reference is made to the interpretation of the *milliampère* meter readings, but no mention is made of the recent researches which indicate that there is an optimum frequency as well as a certain current density and tissue resistance concerned in thoroughly heating a part through and through when this modality is used in medical diathermy.

The section on static electricity deals with the wave, sparks, *effleuve*, induced and simple charges.

The local and general effects than can be obtained by therapeutic radiations, are well described in chapters on phototherapy, heliotherapy, radiant heat and actinotherapy.

The sections on massage, exercise and hydrotherapy will be welcomed by those desiring a brief and practical outline of these valuable methods of treatment.

The author emphasizes the point that all other proved methods of treatment (diet, rest, surgery, drugs, hygiene *et cetera*) must be used when indicated and concludes that there is little doubt that a fuller understanding and use of these physical agents by medical practitioners would give them an improvement in therapeutic results.

The second part of the book is a guide to the physical treatment of the diseases and disabilities that affect the various systems of the body. The technique and charts used in the diagnosis of peripheral nerve lesions are given in attractive form.

The indications for and the technique for the use of the different modalities in the pathological states of each system are adequately described and these chapters, no doubt, will prove a genuine help to many practitioners. In cases in which any particular line of treatment is experimental, the fact is fully acknowledged. Throughout the book all claims for good results are made with moderation. This is particularly noticed in the conclusions drawn from the results of treatment of lobar pneumonia and gonorrhœa by diathermy.

Stewart has apparently had no experience with the ingenious arrangement of electrodes and thermophore made by Siemann and Halske for the treatment of gonorrhœa in the male by diathermy.

We would have appreciated the author's opinion on the value or otherwise of the use of autocondensation in *paralysis agitans*.

In the discussion on the good results obtained in relieving troublesome amputation stumps with the Bardwell whirlpool bath and suitable massage, emphasis is laid on the importance of measures directed to restoring the patient's morale by exercises, organized games and contests.

The book concludes with an interesting and instructive discussion on the management of foot disabilities.

No apologies are made for omitting discussion on X rays and radium from a book bearing the comprehensive title of physiotherapy. Short chapters similar to those dealing with the other agents would be welcome additions to future editions as interest in the therapeutic spectrum scarcely terminates with the ultraviolet radiations.

The illustrations are instructive and the bibliography and index are helpful.

PSYCHOPATHOLOGY.

DR. G. V. HAMILTON is to be congratulated for writing his "Introduction to Objective Psychopathology."[1] Robert M. Yerkes, of Yale University, makes in a brief but eulogistic foreword the significant remark that: "He will presently find himself the leader of a school of psychopathology which will importantly modify our current conceptions of reactive tendencies."

Whether or no his predictions be fulfilled, it is certain that the author has travelled some way on the road towards a rational psychiatry.

The book is divisible into two parts. The first deals with a research extending over a year into the nervous complaints of two hundred patients in a city of thirty-thousand inhabitants on the Mississippi. The place was chosen for survey largely because the people knew nothing of psychoanalysis. The local medical practitioners generously made Dr. Hamilton's "office" a clearing house for their nervous patients.

The author is extremely unconventional in his case notes. His diagnoses include none of the conventional terms such as hysteria, neurasthenia and so forth, but each case is treated on its merits. Examples may be mentioned at random, such as "indirect reaction to inhibited sexual urges," "reaction to need of acquiring status as physically impaired person," "persistent, non-adjustive, affective reaction to baffling business difficulties" *et cetera*.

This behaviouristic method of looking at patients is extremely illuminating, especially when regarded in connexion with animal experimentation.

Dr. Hamilton's patients were by no means all cured and he is engagingly frank about his failures. His treatment consists in inducing his patients to see their behaviour and symptoms in true relief. He shows them how the problem should be solved.

This part of the book closes with a summary of the survey findings. The text is interspersed with shrewd remarks concerning the conduct of his patients and the author makes us regret that he had not the time to treat more.

The second part deals principally with the author's animal experiments numbering some thousands and extending over years. He observes animals, free and in captivity. Mechanisms are devised whereby an animal encounters a situation which it cannot adequately meet by an innate or acquired adjustive mechansm and the results are carefully noted. This is contrasted with the manner in which humans react physically to similar baffling problems. He deals with the foundations of psychology, neural morphology, endocrinology, habit formation, inhibition of responsiveness, unsatisfied major cravings, sexual behaviour *et cetera*. The book closes with a glossary of unfamiliar terms, a bibliography of one hundred and twenty-six titles and an index.

The work can be confidently recommended as worth reading by all who are interested in psychopathology. For a book of this type, it has the advantage of less involved technical English than is often supplied by our American cousins. In any case the happy combination of a biologist and field psychiatrist should lead to the making of interesting comparisons and the reader will not be disappointed.

[1] "An Introduction to Objective Psychopathology," by G. V. Hamilton, M.D., with a foreword by Robert M. Yerkes, Ph.D., LL.D.; 1925. St. Louis: The C. V. Mosby Company Melbourne: Stirling and Company. Royal 8vo., pp. 354. Price: $5·00 net.

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National Insurance in England.

TWENTY-ONE months ago a Royal Commission with Lord Lawrence, of Kingsgate, as chairman was appointed to inquire into the scheme of national health insurance established by the *National Health Insurance Acts*, 1911-1922. Early in March of this year the report of this Commission was issued. It comprises three parts. There is first of all the majority report, signed by nine of the Commissioners. The second part comprises certain reservations which two of the Commissioners who signed the majority report, deemed it necessary to affix to it. The third is the minority report, signed by the remaining four Commissioners.

No attempt will be made to discuss the report as a whole in these columns. Such an undertaking would be of little value to the vast majority of practitioners in Australia, since the Commissioners deal with the scope of the scheme as it exists in Great Britain, with the administration of the several benefits and of the Act itself and with the financial and medical arrangements obtaining at the present time. All this is of interest to sociologists and medical practitioners in Australia as a matter of history. The national health insurance scheme of Great Britain has proved itself to be a success. The insured derive much benefit from its application, although it is admitted that the machine is clumsy, defective in many particulars and in the opinion of some not based on the most advantageous plan. From the report of the Australian Royal Commission on National Health Insurance it would appear that a scheme administered by the friendly societies as "approved societies" with provision for medical benefit is not favoured for the Commonwealth. The medical profession in Great Britain under the old time club practice system was in a totally different position to the medical profession in Australia under the friendly society lodge practice system. The service in Great Britain has improved beyond recognition under the *National Insurance Acts* and the majority of practitioners naturally prefer the present arrangements to the heterogeneous conditions of club practice. It is difficult to form a critical conception of the merits and demerits of the individual details of the scheme without first hand experience of its working. The arrangements are extremely complex; there is apparently a great deal of redundant red tape; post-war industrial difficulties have introduced embarrassing complications of enormous magnitude; the coexistence of financial stringency and the accumulation of large surpluses by the approved societies is an anomaly of political significance. In these circumstances the attention of the profession is directed to the report and members are advised to read the summary of conclusions and recommendations which has been published in *The British Medical Journal* of March 6, 1926 (Supplement, pages 73 to 78), to follow the developments as closely as possible and to await events. It may become advisable to return to this report at a later date, when definite proposals are formulated for a national health insurance scheme for Australia.

In a leading article on the report published in *The Times* on March 3, 1926, it is stated that "it is tne business of the State to care for the health of the community and the duty of the medical profession to regard the health and recovery of the individual patient as the supreme consideration." It is further suggested that "for the establishment of a healthy community the sanitary side may be more efficacious than the medical side of medical science." If these views reflect the considered opinion of sociologists in Great Britain, there would be another reason why the English scheme should not be adopted in Australia. A scheme for the improvement of the health of the community should not be restricted to the granting of monetary benefits during incapacity caused by illness or accident, financial assistance in the case of the death of the bread winner, treatment of persons with limited means during illness and some form of maternity benefit. All these matters are important, but of still greater importance is the enlistment of the active cooperation of the medical profession in the prevention of disease. We claim that the supreme

consideration of the medical profession should be the maintenance of health rather than the attempt to restore lost health and that the direct measures for the control of infections is far more efficacious in the establishment of a healthy community than the development of sanitary science.

Current Comment.

THE WHITE MAN IN THE TROPICS.

THE possibility of the permanent occupation of tropical Australia by a healthy, indigenous white race is a question over which opinions are divided. More than one-third of the Commonwealth lies within the tropics apart from Papua and the Mandated Territory of New Guinea. A Subcommittee was appointed by the Australasian Medical Congress of 1911 to consider not only this possibility, but the conditions conducive or essential to such occupation. A report was presented by the Subcommittee to the Congress held at Brisbane in 1920. The members of the Subcommittee were "unable to find anything pointing to the existence of inherent or insuperable obstacles in the way of the permanent occupation of tropical Australia by a healthy indigenous white race." They considered that the question was one of applied public health "in the modern sense" and they pointed out that the absence of semi-civilized coloured peoples from northern Australia simplified the problem to a considerable extent. They maintained that organization, staff, time and money were necessary for the formulation and carrying out of any scheme of extensive settlement. The report of the Subcommittee was adopted by Congress.

At the Sydney Session of the Pan-Pacific Congress in 1923 several important contributions were made in regard to climate and its effect on human efficiency and its relation to tropical settlement. Professor Griffith Taylor in discussing climatic factors determining tropical settlement said that he regarded as most suited to a white man the climate which attracted him to close settlement. He pointed out that the wet bulb thermometer was the best single instrument for indicating the habitability of any region as regards climate. He concluded that a large proportion of white people would not live in localities of great discomfort until the point of saturation had been reached in more attractive regions. Professor Priestly at the same Congress pointed out that no striking observations had been made of alterations in the physiology of persons in the tropics which could be considered the result of climatic influence. At the same time he laid great stress on "the unhappy lot of women folk" in North Queensland. He referred more particularly to the conditions under which they had to work in carrying out their household and kitchen duties. He regarded the possibility of settling north Queensland with a permanent white population as not proven.

The settlement of the white man in the tropics is the subject of a recent important communication by Dr. R. W. Cilento.[1] The publication is really a survey of facts and figures affecting the residence of white people in the tropics. At the same time Dr. Cilento reports some observations made into house conditions in certain areas. Reference will be made to these later. He begins by pointing out that there is much popular misconception in regard to the tropics. Newspapers, periodicals and retired officials in their reminiscences emphasize unknown dangers and foster a belief that the climate is one of constant heat accompanied by a maximum of discomfort. Danger, heat and discomfort exist as they do everywhere, but in his opinion cannot be regarded as characteristic of the tropics as a whole. In the tropics as elsewhere there are found a series of climates regulated by such well recognized factors as nearness to the ocean, height above sea level, proximity to high mountain ranges, prevailing winds and above all the amount and monthly distribution of the rainfall.

Dr. Cilento contends that life in tropical Australia and in the areas under Australian control offers two distinct problems. The tropical areas of Australia are unique in that they have "no large native population teeming with disease." They are occupied by many thousands of pure-blooded European settlers. Along the eastern coast of Queensland alone these number 103,000. Some of these settlers are of the second and third generation and make up the largest mass of a purely white population settled in any part of the tropical world. They are in reality carrying out a huge and unconscious experiment in acclimatization. The white man in these areas is not the lord of a native race. He is simply a working man, carrying out every occupation from the most laborious tasks to the higher grades of mental effort. The other side of the picture obtains in the tropical dependencies of Australia, Papua and the Mandated Territory of New Guinea. Here the white man is, as he is practically everywhere else in the tropical world, "a more or less exotic bloom." He is the controller and overseer of labour, he is the administrative official and he represents an almost negligible superstratum on the great mass of native life. One of the worst features in the tropics and one of the most difficult with which to deal, is the tendency of the white man in the tropics to regard his residence as of a temporary nature only. In northern Queensland this tendency is gradually becoming less, because the main mass of the population is Queensland born. On the other hand lengthy residence in New Guinea is extremely unusual.

Dr. Cilento devotes considerable space to a description of the climate and geography of tropical Australia and its dependencies. In regard to tropical Australia he confines most of his remarks to north Queensland, because 95% of the residents of tropical Australia are found in that part of the

[1] Service Publication (Tropical Division) Number 7, Commonwealth of Australia, Department of Health; "The White Man in the Tropics, with Especial Reference to Australia and its Dependencies."

continent. He quotes Professor Skertchly, "a strong opponent of white settlement" in the Australian tropics. According to Professor Skertchly the Australian tropic region viewed as a whole enjoys a far more favourable climate than much of the extratropic land and both as regards temperature and rainfall is superior to the regions in the same latitudes in South America and South Africa. The greater part of tropical Australia, the great western plateau, lies over one thousand feet above sea level. A moist tropical climate is the result of heavy rainfall with low elevation. Professor Skertchly regards as unsuitable the area which has an annual rainfall of fifty inches and over. This area extends along the north of Queensland and as far south as the fifteenth degree of latitude. Dr. Cilento points out that this area alone carries a population of many thousands, the overwhelming majority of whom are settled in the hottest and most humid areas. He offers as a sweeping generalization the statement that from Red Scar Bay in the Gulf of Papua, south to the Tropic of Capricorn and west to the borders of Australian control the land is moderately moist to semi-arid; northward to the equator and east to the British Solomons it is moderately moist to exceedingly humid and in both areas there are coastal flats, coastal and inland plateaux and inland steppes and heights.

In regard to the effects of residence in the tropics Dr. Cilento inquires into what physiological changes, if any, occur in white races and what the significance of these changes is. It has been found by Young that while the body is at rest its temperature does not vary appreciably from that which characterizes it in temperate zones. The temperature rises more quickly after exercise in tropical zones and subsequently decreases at a slower rate. This is more evident in the newcomer who is not yet acclimatized. In regard to skin colour a black skin is of advantage in that by absorption of heat the body is raised more readily to the sweating point and so to the relief which is experienced at that stage. Of far more importance to the comfort of the person than the actual degree of external temperature or even of humidity is the degree of heat stagnation. With high external temperatures the heat lost by sweat evaporation is the most important factor regulating body temperatures and this heat loss is proportionate to the amount of air movement. For further information on this aspect of the question the reader is referred to an article by Corlette, published in THE MEDICAL JOURNAL OF AUSTRALIA of February 17, 1923, and to the observations of C. J. Martin and others at the Pan-Pacific Congress, recorded in our issue of September 29, 1923. The sense of discomfort experienced by Europeans in the tropics is largely a matter of clothes and house ventilation. Rubner pointed out that there is a layer of stagnant air enclosed between the skin and the clothes. The air layer is charged with heat, with moisture and with the exhalations of the skin and is at all times at a much greater temperature than the external air. For this reason a person wearing close-fitting clothes, whether he be in the tropics or in a temperate climate, lives always under tropical conditions as far as his clothed body is concerned.

The statement is commonly made that life in the tropics leads to an alteration in the blood and it is not surprising that the lay mind accepts this statement without question. The first thing which strikes a visitor to north Queensland from the south, is the extreme pallor of the women and children. Priestly and Breinl made an examination of 574 apparently healthy school children ranging from seven to fifteen years of age (see THE MEDICAL JOURNAL OF AUSTRALIA, September 29, 1923). The only abnormality discovered was a slight increase in the number of leucocytes. There was no anæmia. Young, Breinl, Harris and Osborne found a small, but variable increase in blood solids after profuse sweating in Townsville and Breinl on investigating the effect of high wet bulb temperature on the blood pressure of labourers on the wharves in Townsville failed to find results differing materially from those obtainable during strenuous work in temperate climates (THE MEDICAL JOURNAL OF AUSTRALIA, April 16, 1921). Dr. Cilento points out that many cases of anæmia in the tropics are due to malarial and helminthic infections and that the common pallor of the healthy tropical resident is due to a tropical ischæmia or a heaping up of the surface epithelium which is of no real significance save as an adaptive protection. Ruddiness in an old tropical resident generally indicates ill-health or alcoholism. Sundström found that there was a general deficiency in the organic and lipoid fractions of the phosphorus containing constituents of the blood and that this was more definite in women than in men. He also found a definite alkalotic blood condition. This is possibly productive of the easy fatigue and depression of the tropics and may be neutralized by muscular effort.

In regard to the digestive and secretory systems, Dr. Cilento refers to the description given by Manson-Bahr of the condition known as tropical liver. This begins as a purely physiological condition, a condition of hyperæmia from functional inactivity. When physiological hyperæmia is pushed a step further, congestion and blood stasis result. Climate is obviously a very indirect cause of all this morbidity and Dr. Cilento points out that faulty habits of living may produce it in any climate.

In regard to the generative functions and growth Dr. Cilento finds some justification for the statement that in both male and female there is greater generative vigour in the tropics and that excess in venery is more speedily followed by exhaustion and neurasthenia than is the case in temperate climates. At the same time he holds that this is not consequent on any climatic factor, but is related to excess of opportunity and to the absence of the restraining influences of civilized life and environment. He does not find confirmation of the statement that puberty occurs at an earlier age in the tropics. Acceleration of puberty depends in individual instances more upon better feeding with accelerated general development, increased opportunity for

sexual enlightenment and speculation and an atmosphere of licentiousness than on actual climatic conditions. In regard to growth the stimulating effect of tropical conditions on the body is important. Sundström found on a careful examination of one hundred growing girls in Townsville that growth and development were most definite and satisfactory during the time when heat and humidity were greatest.

Reference has already been made to Priestly's views on the conditions under which women are compelled to work in north Queensland. Dr. Cilento refers to the so-called tropical neurasthenia which is commonly regarded as being due to climate, and also to what Cumpston has described as the "kitchen neurasthenia" of the north of Australia. Apart from the neurasthenia due to definite organic cause, two types of neurasthenia are seen in the tropics under Australian control. There is a neurasthenia due to indolence and self-indulgence and there is also a neurasthenia due to the depressing monotony of hard work under extremely trying circumstances. From what has already been said it will be obvious that the former is more common in New Guinea and Papua and the latter in tropical Australia proper. Neurasthenia in the tropics manifests itself as an instability of the mental equilibrium, fits of exuberance alternate with states of depression, unwarrantable irritability is manifested over trivial matters and bursts of fury especially in male persons are not uncommon. Associated with these symptoms is a diminution of mental ability, activity and power of concentration and to this are often added a lack of self-confidence and an unresponsive memory. Dr. Cilento points out that in north Queensland as opposed to New Guinea neurasthenics constitute a mere trifle of the total resident population and the indications of neurasthenia are as a rule mild, manifesting themselves as an inability for concentrated effort, a tendency to inertia and a revolt against circumstances. Here he asks two very pertinent questions: "Is neurasthenia from social and environmental circumstances confined to Europeans in the tropics?" and "Is it confined to the tropics and can climate *per se* be regarded as anything but a contributory factor?" He holds that the answer in each instance must be in the regative.

In connexion with neurasthenia Dr. Cilento discusses the conditions under which women are compelled to live. He draws a telling word picture of the life of women in the tropics where there is a subject native race. He shows that it is regarded as undignified for her to attend personally to any of the duties of the household, that she is alone for a large part of the day and that even her children, if she consents to have any, are left to the care of native nurses. In Australia the position is very much otherwise. There is no large native population. Help in the home is unobtainable or obtainable only at a figure which is beyond the reach of the large majority. Every type of manual labour and "menial service" must be done by the white man and woman of the community, just as it is in any other portion of the white civilized world. Dr. Cilento holds strongly that this is the happiest augury for the future of a "white Australia." Sundström demonstrated in Townsville that those people who most nearly approached what is regarded the standard for temperate climates, were those engaged in hard work. A most significant finding was obtained in the person of the caretaker of the Australian Institute of Tropical Medicine. She was found to reach a perfect standard of normality after she had spent two hours on a muggy tropical morning cleaning the Institute. The least satisfactory result of all tests was that taken from a girl who followed the sedentary occupation of stenographer and who avoided every form of exercise.

Dr. Cilento also makes reference to the vital statistics of Queensland as compared with the rest of the Commonwealth. Space does not permit adequate discussion of this aspect in this place. It must suffice to point out that Dr. Cilento holds that they compare more than favourably with the latter. He states that the birth rate statistics do not vary in the north from those obtaining in the south of Queensland. He draws two important conclusions. The first of these is that physical exercise, work, is absolutely essential for life in the tropics and the second is that preventive medicine must be a foremost factor in every aspect of life and industry.

Dr. Cilento also reports observations made into the effect and result of tropical residence in north Queensland. This included a thorough investigation of the sociological and environmental conditions of the people in three sets of towns. These were chosen as representing the hot, moist coastal area, the coastal plateau and the hot, dry inland climates. Townsville and Cairns represented the first, Atherton, Chillagoe and Charters Towers represented the second and Julia Creek and Cloncurry the third area. The results of this investigation provide very interesting reading. It is impossible to give the findings in full. In general the houses are small and not well suited to the climate. Though kitchen stoves are generally built in a recess, the ventilation in approximately 50% of kitchens is bad. Houses as a rule are badly planned and even water is not generally laid on so that it can be obtained conveniently for household purposes. Relatively few women have any knowledge of household economy or sanitation and an equally small number take trouble in attempting to arrange their hours of work so that they may have time for rest. Dr. Cilento discusses the statement frequently made that while women may live for short periods in the tropics without suffering permanent harm, yet they cannot bear healthy children and that their fertility is lessened. As a result of his investigation he holds that this view is entirely disproved. He draws attention to the fact that a considerable second generation of tropically born children is rapidly springing up in Queensland.

The question of a white Australia has always been in a large measure political. It is obvious that apart from politics the question is of the utmost importance to Australia and Australians. If the northern part of Australia can be kept racially

pure and if industries can be made to thrive in every part of the continent, the nation will be the gainer not only in commercial prosperity, but in the type of individual that can be produced. The northern regions are producing a hard and wiry type of man who is capable of great endurance. Dr. Cilento has had great opportunities of observing the north and its people. He has made out a good case for the permanent occupation of the tropics by a white race. He has demonstrated the possibility and indeed this may be taken for granted, for, as he points out, it is actually being done. At the same time it is evident that life in a humid atmosphere is uncomfortable when compared with life in other places. The north must be made more habitable and attractive. Medical observers will agree with Dr. Cilento that the question is largely one of preventive medicine. Malaria, helminthiasis and other diseases are found in the Australian tropics. They can be dealt with and eventually controlled. More than this, however, is necessary. The general principles of preventive medicine must be applied. The question need not be political, but it is here that governments are necessary. Preventive medicine and economic improvements require money. The money would be well spent, it would be returned many fold. Dr. Cilento has drawn up a list of what he terms essentials. It is a formidable one. He classifies them under four headings: Facilities for import and export, food, sanitation and development and preventive measures. In regard to the first of these he points out that adequate means of municipal communication and of communication between various centres are necessary as well as the provision of facilities for the import of necessities and the export of production. In regard to food three things are required: the provision of a water supply, adequate both in quantity and in quality, the provision of a sufficient milk supply and the provision of a plentiful supply of fresh vegetables and fruits. Under the heading of sanitation and development he includes suitably constructed houses and business accommodation, competent measures for the disposal of waste, both domestic kitchen refuse and fæcal material, properly drained streets, the provision of hospital facilities and the provision of educational opportunities. The preventive measures advocated by him include protection against insect-borne diseases by the constant reduction of mosquitoes, flies, rats and other disease-bearing creatures, as well as the prevention of the influx of diseases from without and their spread within the locality. He points out that in the majority of the towns in north Queensland practically the whole of these essentials are wanting at present, though conditions are improved from day to day.

HYPERTONIC SALT SOLUTION IN THE TREATMENT OF ACUTE POLIOMYELITIS.

WEED and MCKIBBEN in 1919 showed that the intravenous injection of hypertonic salt solution in animals produced a decrease in the size of the brain and a fall in the pressure of the cerebro-spinal fluid. Cushing and Foley applied these findings to the surgical treatment of cerebral herniæ and found that the reduction produced in the size of the herniæ made operation easier of performance. Barach, Mason and Jones in 1922 found that the immediate result of an injection of hypertonic saline solution was an increase in the volume of the blood plasma. Osmosis produced in this way has been used in connexion with other pathological conditions. Aycock and Amoss in 1923 reported the recovery of a patient who was *in extremis* owing to severe poliomyelitis, after they had given an intravenous injection of hypertonic saline solution and an intrathecal injection of serum from a convalescent patient.

Dr. T. Cook Smith has recently used this method in the treatment of a boy, aged five years, who was so ill that in his opinion recovery seemed almost impossible.[1] He points out that morbid anatomists have described an inflammatory œdema surrounding those areas of the brain which are the chief seat of injury in acute poliomyelitis. He regards it as not unreasonable to suppose that the resulting pressure is in large measure responsible for the paralyses which occur in the acute stage of the disease, but which disappear later on. He expresses the opinion that if the pressure resulting from the inflammation can be diminished at the critical moment of the disease, the acute situation may be successfully passed and the life of the patient saved.

Dr. Smith describes the history of his patient. On the day before admission he was tender on being touched anywhere. On admission he was in a state of extreme apathy and stupor. The temperature was 38.3° C. (101° F.), his pulse rate was 120 and his respirations numbered 40 in the minute. Respiration was purely diaphragmatic. The neck was stiff, but Kernig's sign was absent. Weakness of both extremities was noted. The boy was too ill to undergo lumbar puncture. A diagnosis of acute poliomyelitis with respiratory paralysis was made. The patient then became cyanotic. Oxygen, caffeine and atropine were administered. Respiration became confined to the left side of the diaphragm and the patient appeared to be dying. Subsequently forty cubic centimetres of a 30% solution of sodium chloride were injected into the right femoral vein. Pallor and quickening of the pulse occurred and respiration ceased. After a few moments respiration began again and after nine hours the administration of oxygen was discontinued. Two days later Kernig's sign became manifest and after this improvement gradually occurred. Subsequently the *recti-abdominis* muscles, the intercostals and the right *tibialis anticus* remained paralysed.

Dr. Smith points out that recovery may have been coincident with the treatment adopted, but in his opinion the treatment was responsible for recovery. He indicates that he is in no position to define the optimum concentration or dosage of saline solution, but holds that in a similar case this form of treatment should be tried.

[1] *American Journal of Diseases of Children*, March, 1926.

Abstracts from Current Medical Literature.

SURGERY.

Carcinoma of the Prostate.

A. CLIFFORD MORSON (*The Lancet*, February 13, 1926) discusses briefly the technique of operation for carcinoma of the prostate. The author has devised an operation which according to him differs in no material respect from that of Young, of Baltimore, except that the author gains access to the growth by the suprapubic route while Young selects the perineal route. After briefly reviewing the anatomy of the region and laying stress on the importance of the prostato-peritoneal fascia or fascia of Denon Villiers, the author goes on to describe the mode of spread of prostatic cancer. The cancer cells spread out in a fan-like manner beneath the bladder from the parent mass; they pass above lymphatic channels which are buried in fibrous tissue enveloping the *vasa deferentia* and the seminal vesicles. The progress of the malignant cells is arrested at the junction of this fibrous tissue with the peritoneum of the pouch of Douglas. This acts as a barrier and diverts the spread of the cancerous material to the ureters which it embraces and causes to become stenosed. The operation may be divided into two stages, the first stage consisting of the separation of the prostate from the surrounding structures and the second comprising the dissection and removal of all the tissues which form the base of the bladder as far back as the inter-ureteric bar and the anterior wall of the rectum. The best light is obtained by the use of a retractor carrying an electric light bulb into the interior of the bladder. It is essential to use the Trendelenburg position in order to obtain a good exposure of the internal meatus and the bladder base. An incision is made round the internal meatus and with a blunt dissector the line of cleavage between the growth and the fibrous capsule is determined. Should the base of the bladder be involved, the incision should include a triangular area from the ureteric orifices to the internal meatus. Partial mobilization having been obtained, the mass is pulled into the bladder with the attachments to the base of the bladder and the seminal vesicles still intact. The second stage of the operation consists in the removal of all tissues anterior to the front of the rectum including the mucous membrane of the bladder, if this is considered to be invaded by the disease. The fascia and seminal vesicles are stripped from the loose fatty tissue which separates them from the bowel. The *vas deferens* is divided close to the brim of the pelvis and the mass is further separated from the bowel. Lastly the whole mass is removed by division of the fascia of Denon Villiers at its attachment to the peritoneum at the bottom of the pouch of Douglas. It is not a serious matter to buttonhole the peritoneum, provided the opening is closed immediately, but buttonholing of the rectum, leading to recto-vesical fistula, must be avoided at all costs. The operation is completed by the insertion of a tube into the bladder. The immediate after treatment is the same as that of any other form of prostatectomy. The author claims good results from the operation and states that many patients may thus be saved who would otherwise die.

Carcinoma of the Colon.

KATE PATERSON BROWN (*Edinburgh Medical Journal*, January, 1926) analyses the histories of one hundred and seventy-one patients treated for carcinoma of the colon at the Royal Infirmary, Edinburgh, during the past ten years. It has been possible to trace the end results in a considerable proportion of the cases and thus to compare the value of different forms of operation. The highest incidence of the disease (ninety-one cases) was in the pelvic colon; the caecum was involved in twenty-two instances, the ascending colon and hepatic flexure in thirteen, the transverse colon in fifteen, the splenic flexure in nineteen and the descending colon in eleven. The average age of patients was about fifty-five years. The early symptoms may easily be overlooked. Pain is rarely a prominent symptom; it is associated generally with irregularity in the action of the bowel, constipation frequently alternating with diarrhœa. When blood is found in the stool with mucus and when loss of weight has occurred and when anæmia is present with or without a palpable tumour, the diagnosis is almost certain. Signs of obstruction are found only in the advanced stages of the disease. The author groups treatment under five headings. The first is the one stage operation. Under the second heading is placed the two stage operation which consists in bringing the tumour out of the abdomen and resection and anastomosis at a later date. The third form of treatment consists in resection and caecostomy carried out either before or at the time of resection. The fourth type of treatment is the three stage operation of Mikulicz. The last form consists in preliminary anastomosis followed by resection. The first is eminently satisfactory for proximal, but not for distal colon tumours. In the second form of treatment 50% of deaths occur, as the second operation has necessarily to be performed in an area contaminated by fæcal discharge. In the third group the mortality was only 21·7%, a definite improvement of that of the previous group. The cases in both these groups were characterized by obstruction. The fourth group was attended by a mortality of 25%, a figure which the author regards as higher than that usually accompanying this form of operation. Preliminary anastomosis is of most value in the treatment of tumours of the proximal part of the colon and is most suitable for a two-stage operation on a tumour in this region. The author lays stress on the value of caecostomy in association with colon carcinomata. For inoperable tumours of the proximal part of the colon an ileo-colostomy is a safe and satisfactory operation and it gives the patient greater comfort than a permanent caecostomy. The author records the fact that of one hundred and seventy-one patients suffering from carcinoma of the colon who were admitted to hospital, forty-three were suffering from an acute intestinal obstruction. In connexion with these figures the author again emphasizes the value of caecostomy as a life saving measure. The end results as a rule are disappointing. A considerable proportion of the patients in the author's series died within two or three years of operation and in this list of cases spread over ten years the longest period of survival was four years. Considered from the point of view of site caecal tumours yielded the best results. The type of palliative operation performed appeared to have little effect on the course of the disease. The author finds that the patient generally succumbs within a year or eighteen months irrespective of whether anastomosis or colostomy is performed.

Pain in the Right Iliac Fossa.

H. W. CARSON (*The Lancet*, February 13, 1926) reviews the commoner causes of pain in the right iliac fossa. The late Frederick Treeves first conceived and taught the existence of chronic appendicitis. The author holds that many patients complain of chronic pain in the right iliac fossa which is not cured by removal of the appendix and which is really not due to chronic appendicitis at all. One of the most common causes of pain in this region is muscle strain. This is seen most often in two classes of patients, firstly the young school girl who strains her muscles at sport and secondly, an athletic man who is developing a hernia due to putting on weight. A second frequent cause of right iliac pain is a lesion in the urinary tract; the most common abnormality is a stone in the ureter. In this connexion the author refers to an American writer who declared that 25% of patients with ureteral calculi undergo appendicectomy before the true cause of the pain is discovered. Tuberculous mesenteric glands also lead to errors in diagnosis as also may tumours of the colon whether tuberculous or malignant. Pain in the right iliac fossa may even be due to malignant disease in the left colon. In these circumstances back pressure causes distension of the caecum. Malignant disease of the descending colon should be suspected in a patient over fifty years of age who complains of these symptoms. Passing over causes such as colitis, spinal disease, *tabes dorsalis* and tumours

of the spinal cord, the author discusses the obscure cases of vague pain in the right iliac fossa which are not due to any of the above causes. Such pain is found in spare women of from twenty to forty years of age with narrow chests and reduced pelvic outlets who are the subjects of general abdominal ptosis. The cause of the pain is yet to be determined as neither nephropexy nor removal of the appendix, nor division of bands (Jackson's membrane) nor colopexy seems of any avail. No cure has yet been found for this pain. X ray diagnosis of chronic appendicitis is regarded by the author with suspicion and as of little value. In conclusion he expresses the opinion that pain confined to the right iliac fossa should not be considered as due to appendicitis, especially in the absence of a previous severe attack, until all other possibilities have been excluded by the most painstaking investigation.

Tuberculous Tumour of the Caecum.

CLARENCE A. JOHNSON (*California and Western Medicine*, February, 1926) reports a case of tuberculous tumour of the caecum. This lesion is of rare occurrence. Of all cases of tuberculous involvement of the intestine only 8% occur in the caecum. Primary infection is rare in adults, but secondary infection in the late stages of pulmonary tuberculosis is more common. Infection spreading to the lumen of the gut from the peritoneum or from tuberculous glands in the mesentery is quite exceptional. The tuberculous ulceration generally begins in the Peyer's patches and the gut becomes swollen and thickened around the bases of the ulcers. Tuberculosis of the caecum occurs in two forms, an ulcerated and a hyperplastic form. The latter condition resembles a new growth in causing stenosis. The usual symptoms are stated to be alternating diarrhœa and constipation with occult or evident blood in the stools. The swelling is generally palpable, hard and sausage shaped. Obstructive symptoms supervene and if operation is not undertaken, death usually results in one to three years. The condition must be distinguished from appendicitis, actinomycosis and malignant growth. In ulcerative forms which occur as terminal complications of advanced pulmonary tuberculosis, there is little possibility of treatment by surgical means. For hyperplastic tumours complete extirpation with resection of the glands as far back as the origin of the ileocolic artery is advised. About twenty centimetres of the ileum should be resected together with the ascending colon up to the junction of its middle with its upper third; this is followed by a lateral or end to end anastomosis.

Experimental Hydronephrosis.

FRANK HINMAN (*Surgery, Gynecology and Obstetrics*, February, 1926) has carried out work in experimental hydronephrosis and has noted the important part that arterial changes bear to the pathological alteration of the kidney. Rabbits were used for the experiments. It was found that the higher the ligature was placed on the ureter, the quicker the changes occurred. A very careful technique was followed in injecting the arterial circulation of the kidney. There are four zones in the kidney, namely the subcapsular, the cortical, the corticomedullary and the medullary. There is a particular arrangement of vessels for each of these zones. The renal circulation pursues two directions in relation to the cavity of the pelvis, circumferential and radial. The main subdivisions of the renal artery pass round circumferentially and the finer branches are distributed in a radial direction. With the production of hydronephrosis the arterial circulation undergoes two phases of alteration. In the beginning there is short period of apparently purely mechanical interference and then there supervenes a second phase in which there is an actual alteration in the length and calibre of the vessels. Since arteries are elastic tubes, they become stretched and more attenuated as the pelvis and kidney expand and hence their lumina become proportionally narrower. There ensues a reduced flow of blood through these channels, leading to a state of ischæmia. This state by lowering tissue tone favours the progress of atrophy to its ultimate completion. It is thus evident that the progressive dilatation of the pelvis and kidney is much assisted by vascular changes.

Muscle and Fascia Suture with Relation to Hernia Repair.

A. R. KOONTZ (*Surgery, Gynecology and Obstetrics*, February, 1926) discusses the question of muscle and fascia suture with special relation to hernia repair. The percentage of recurrences of hernia varies so widely, that it must be due to different methods of tracing and following patients. However, the figure remains too high. In all operations for inguinal hernia one of the chief requisites for a cure is the effective repair of the faulty abdominal wall. There is much difference of opinion as to whether the internal oblique and conjoined tendon actually unites with Poupart's ligament. Seling and Chouke recently stated that normal muscle will not unite firmly with fascia or ligament. Fascia will unite with fascia. In experiments with thirty-seven dogs, the classical operations on the inguinal canal were performed with different types of suture and various materials. The animals were examined at periods varying from one week to nine months. It was found that in spite of considerable tension the internal oblique muscle united and remained united with the inguinal ligament in a firm state, but microscopical examination showed that the union was between the fascia of the ligament and the connective tissue of the epimysium. Hence it is in effect a fascia to fascia union. It was found that surfaces placed in contact must be completely deprived of areolar tissue, otherwise the strength of the union is very slight. Such surfaces should be thoroughly scraped and scarified to obtain the strongest union. In the case of aponeuroses and deep fascia the edges should be overlapped and the surfaces denuded thoroughly of areolar tissue. The larger the area in contact, the firmer the union. The cutting away of a small strip of the edge of the internal oblique and the forming of a raw surface tends to make the union firmer than usual. If these precautions are taken, it appears that a good union can be assured.

Traumatic Neuritis of the Ulnar Nerve.

HARRY PLATT (*The British Journal of Surgery*, January, 1926) writes of the pathogenesis and treatment of traumatic neuritis of the ulnar nerve in the post-condylar groove. The nerve is here in a position of extreme vulnerability. Involvement of the ulnar nerve is a rare complication in recent fractures of the lower end of the humerus. The common type of fracture to be accompanied by a nerve lesion is separation of the internal condyle. The lesion is usually a primary contusion, a physiological block without loss of continuity. The symptoms are slight and rapid spontaneous recovery tends to occur. A secondary ulnar neuritis may be an unavoidable sequel perhaps of injudicious treatment, such as forcible passive movements in the first few weeks. In all cases of severe and persistent neuritis the nerve trunk should be explored and displaced to a new bed in front of the condyle. Late involvement of the ulnar nerve (delayed ulnar palsy) is a characteristic remote sequela of certain fractures of the external condyle sustained in childhood. There are three periods, the fracture, a long latent period, rarely less than ten years and the period of the development of neuritis. It is a friction or tension neuritis due to the *cubitus valgus* deformity. The nerve is forced to take a longer course. Operative measures are essential in these cases. It is necessary to shorten the course of the nerve. Two procedures are worthy of adoption. The most suitable is anterior transposition of the nerve and the other is that of supra-condylar osteotomy. Minor degrees of hypermobility of the ulnar nerve are often discovered in the routine examination of the elbow region, but a few individuals develope a true complete dislocation of the nerve from its groove. Variations in the depth of the groove and congenital or acquired laxity of the nerve sheath or arcuate ligament and more especially an exaggerated carrying angle are all possible explanations. The onset of neuritis in such a movable nerve may be long delayed and when it does occur, is of the friction type, with the ultimate formation of a nerve spindle—fusiform neuroma. There is a simple and effective operation which is universally applicable in all cases and that is anterior displacement of the nerve trunk.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SECTION OF NEUROLOGY AND PSYCHIATRY AND THE SECTION OF PATHOLOGY OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on October 15, 1925, at the Medical School, University of Sydney, DR. A. W. CAMPBELL in the chair.

Trophic Glial States.

DR. OLIVER LATHAM gave a demonstration on "Trophic Glial States in Spinal Cord Lesions" (see page 507).

The lecture concluded with an epidiascope demonstration of sections of spinal cords from cases of simple and malignant intramedullary tumours, extracordal tumours, Friedreich's ataxia, ataxic paraplegia, Erb's form of spinal syphilis, pernicious anæmia, Addison's anæmia, primary lateral sclerosis, *tabes dorsalis*, disseminated sclerosis and primary degeneration of the neurone. In some of the sections evidence of glial atrophy was to be seen.

PROFESSOR A. E. MILLS congratulated Dr. Latham on his paper and his demonstrations. In the course of his remarks he pointed out that apparently the nutrition of all tissues was dependent upon the trophic influence of certain neurones. He cited the effects resulting from poliomyelitis and suggested that it was probable that in this disease not only were the cells of the ventral horn of the cord affected, but also the cells of the lateral horn. It might well be the case that the trophic cells of the cord were situated in the lateral horns. The question then arose whether the nutrition of the nervous system and its extensions was influenced in a similar manner, namely, by these same trophic cells. Such a view, he thought, was highly probable, but it was extremely difficult to bring forth proof of it.

With regard to the changes in the cord in tabes and in combined degeneration of the cord apparently the pathological histology was the same, but due to different causal agents, in the one syphilitic toxins, in the other some toxin of unknown origin frequently associated with but not necessarily due to pernicious anæmia and its attendant achlorhydria. Hurst's statements on the relation of achlorhydria to combined degeneration were very suggestive, but were, the speaker thought, too sweeping, especially as a condition of achlorhydria was to be found in a certain small percentage of apparently normal healthy people.

PROFESSOR A. N. BURKITT asked the question whether trophic nerves could actually be seen. There were great difficulties in studying nervous tissue. Sympathetic fibres were so fine that only the highest magnification and favourable staining would enable them to be seen. Dr. Wilkinson had recently shown nervous fibres which would appear to be proceeding to fat cells and which might be affecting fat metabolism. It had been shown that injury to the base of the third ventricle led to the deposition of fat without the pituitary gland being affected and this suggested that there was a higher nervous arc functioning through the sympathetic nervous system to fat cells, glands, epithelium and perhaps to nervous tissue itself. Pavlov and his workers suggested the possibility of trophic nerves. Definite anatomical and physiological as well as clinical evidence was accumulating in favour of a trophic or regulatory nerve supply to tissues and organs in which their existence had hitherto been unsuspected, and Dr. Latham was to be congratulated in bringing forward further evidence in this direction.

DR. A. W. CAMPBELL said that Dr. Latham's paper had brought forward much evidence that the nervous system contained a considerable amount of sympathetic nervous material. In staining nervous tissues by ordinary methods all the supporting structure was wiped out and therefore microscopical examination was often somewhat misleading. Dr. Latham had referred to his (Dr. Campbell's) investigations with Dr. Henry Head regarding the changes in the nervous system in *herpes zoster*. In these investigations the posterior root ganglia in monkeys were exposed and chromic acid was injected into them. Herpes did not result from such injections and it was assumed that in addition to mechanical injury a virus was necessary. The changes in the cord in combined degeneration seemed to Dr. Campbell to be of vascular origin. Dr. Campbell congratulated the reader of the paper on the work he had done.

Dr. Latham, in reply, said it was often impossible to distinguish parts of the lesions taken from cases of combined sclerosis from those of *tabes dorsalis*, but an examination of the whole cord in either case would establish a diagnosis. In combined sclerosis the root zone escaped, but this region was most commonly picked out in tabes.

Digestion and Mental Disease.

DR. J. BOSTOCK read a paper entitled: "Digestion and Mental Disease: An Analysis of One Hundred Consecutive Fractional Test Meals, with Some Animal Experiments" (see page 510).

DR. F. S. HANSMAN in discussing this paper said that there was still much to be found out as regards the significance of gastric findings. He wished to bring forward certain phenomena for explanation.

Firstly, while taking samples in certain cases it was difficult during an appreciable time to aspirate any fluid, even though more tube was swallowed or portion of the tube was withdrawn and though air could be easily forced into the stomach and then suddenly there was a squirt of fluid almost sufficient to raise the piston of the syringe.

Next, why did the stomach empty so quickly in achlorhydrias in which there was no acid, and in duodenal ulcer in which there was a large amount of acid, whereas in other cases of hyperacidity there might be a highly acid residue in the stomach at the end of three hours?

Lastly, when was the proper time to give alkalis therapeutically, during the digestive cycles, when acid probably tended to be secreted as it was neutralized or between digestive cycles to neutralize acid remaining in the stomach?

DR. CYRIL SHEARMAN offered his congratulations to Dr. Bostock. He thought it regrettable that it had not been possible to carry out total chloride estimation in all the series as estimation of the acid content alone did not give a true indication of the secretion of acid in view of possible neutralization by reflux from the duodenum.

Further interest was added to Dr. Bostock's investigation in view of any possible analogy between the association of deficient hydrochloric acid secretion with the manifestation of nervous lesions and the conditions met with in certain cases of Addison's anæmia where a combined sclerosis was associated with a total achlorhydria.

PROFESSOR A. E. MILLS said that much care should be exercised before any general conclusions were drawn from variations in the hydrochloric acid content of the gastric juice. He pointed out that some people secreted more tears, more mucus, more sweat than others and that these differences in secretion expressed in a way the make-up of different individuals. It was pleasing to note that, though Dr. Bostock had done considerable work and had obtained some suggestive results, he did not rashly jump to conclusions. Professor Mills offered Dr. Bostock his hearty congratulations on a tedious piece of work carried out in a careful way.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Broughton, John Walter, M.B., Ch.M., 1924 (Univ. Sydney), Murdoch Street, Cremorne.

Dearberg, John Louis, L.R.C.P., L.R.C.S. (Edinburgh), 1926, L.F.P.S. (Glasgow), 1926, 133, Oberon Street, Coogee.

Downes, Clement James, M.B., Ch.M., 1923 (Univ. Sydney), 9, Dickson Street, Haberfield.

Francis, Neil Whinney, M.B., Ch.M., 1926 (Univ. Sydney), 12, Sutherland Road, Chatswood.
Goldrick, Vincent Thomas, M.B., Ch.M., 1925 (Univ. Sydney), "Carisbrooke," Springfield Avenue, Darlinghurst.
Hay, Vivian Charles Langtree, M.B., Ch.M., 1925 (Univ. Sydney), Kirribilli Avenue, Kirribilli.
Hardie, John Patrick, M.B., Ch.M., 1925 (Univ. Sydney), 41, Fern Street, Randwick.
Laidley, John Walwyn Shepheard, M.B., Ch.M., 1924 (Univ. Sydney), Buckhurst Avenue, Double Bay.
Hungerford, Doreen, M.B., Ch.M., 1926 (Univ. Sydney), "Glanworth," Chandos Street, Ashfield.
MacCallum, Walter Paton, M.B., Ch.M., 1924 (Univ. Sydney), Wyuna Road, Point Piper.
Macindoe, Norman Macleod, M.B., Ch.M., 1925 (Univ. Sydney), Orange.
Macpherson, James Simson, C.M.G., L.R.C.P. (Edinburgh), 1886, L.R.C.S. (Edinburgh), 1886, L.F.P.S. (Glasgow), 1886, Red Hill, Burrowa.
Maitland, Herbert Lethington Chisholm, M.B., Ch.M. 1923 (Univ. Sydney), 147, Macquarie Street, Sydney.
Morris, George Bentham, M.B., Ch.M., 1925 (Univ. Sydney), 31, Woodstock Street, Waverley.
Salter, Arthur Edward Harrison, M.B., Ch.M., 1925 (Univ. Sydney), Abbotsford Road, Homebush.
McCarthy, Edward Francis, M.B., Ch.M., 1921 (Univ. Sydney), c.o. H. Sparkes, Esquire, Harbourne Street, South Kensington.
Nolan, Geoffrey Russell, M.B., Ch.M., 1924 (Univ. Sydney), Belmont Avenue, Wollstonecraft.
Steele, Spencer Carlisle, M.B., 1925 (Univ. Sydney), Norfolk Road, Epping.
Wilcox, Rita, M.B., Ch.M., 1926 (Univ. Sydney), Saint Neot Avenue, Potts Point.
Waterhouse, Arthur Stanley, M.B., 1924 (Univ. Sydney), Torrington Road, Strathfield.

Medical Societies.

THE SYDNEY HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE SYDNEY HOSPITAL CLINICAL SOCIETY was held in the Maitland Lecture Theatre, Sydney Hospital, on October 22, 1925, DR. T. FIASCHI, D.S.O., the President, in the chair.

Chronic Chorea.

DR. G. C. WILLCOCKS, O.B.E., M.C., showed a male patient, aged forty-three years, a labourer, who complained that he had been very nervous for about six years. During the previous few months he had had difficulty in walking and had been unable to control the movements of the hands and legs. On examination the patient was seen to be thin, rather pale and worn looking. He walked with a swaying gait. Constant irregular movements of the face, arms and legs were present. During conversation he continually made grimaces. His speech was interrupted but not slurred. When asked to show his tongue he protruded it suddenly to its full extent and withdrew it with equal suddenness. The movements of the face, arms and legs were irregular in time and direction. The same movement was not repeated. The cranial nerves appeared normal, no abnormality of the *fundus oculi* was present. Muscular power was good and no wasting of the muscles was present. The reflexes were not affected. Sensation was normal. Mentally the patient showed signs of defect which were enhanced by the disorder of speech. His condition was one of early dementia. He could not be relied upon to carry out simple tasks. He forgot in a few moments what he was told. His memory and intelligence were such that he was not considered capable of looking after himself. The blood serum had been submitted to the Wassermann test, but no reaction had occurred. The condition was typical of chronic chorea.

Dr. Willcocks said that the classification of chorea of this type was difficult. Huntingdon's chorea commenced between thirty and forty years of age and was familial. Senile chorea was said to begin at about fifty years of age and to progress less often to dementia. There was, however, no hard and fast rule and the conditions were often indistinguishable except by the family history. The cause was unknown. Morbid anatomy of the very varied kind had been described. Pfeiffer's description of an increase of glial cells and fibres and thickening of the vessel walls in the cortex, *corpora striata* and thalami appeared to fit in with the present ideas of the functions of those parts.

Hysteria.

Dr. Willcocks also showed a male patient, aged twenty-five years, who had presented himself at the Out-Patient Department six weeks previously with a complaint of numbness and tingling in the hands. He had stated that he came into contact with arsenic in the course of his work. On examination no paresis or sensory disturbance had been detected and the reflexes had been normal. The urine had been sent to the Pathological Department for examination. A report had been received that it contained arsenic. Two weeks later the patient suffered apparently from definite paresis of the forearms, especially the extensor muscles. On shaking hands with him, however, it had been noticed that the grip seemed unduly weak considering the state of his muscles. Further examination had revealed the fact that the patient resisted certain movements when asked to raise his arms. He had been admitted to the wards.

No sensory changes had been detected. The reflexes and muscle reactions to electric stimulation had been normal. The fæces were reported to have contained arsenic. After a few days the patient had recovered the power in his hand and forearm and had been discharged from hospital. The resident medical officer had ascertained that another man working at the same place had suffered from arsenical neuritis.

Dr. Willcocks said that it was certain that the apparent paralysis of the forearm was not due to neuritis, for it recovered in a few days. Moreover, the reflexes had been unaffected and no sensory changes had been detected. The subjective sensations of which the patient had complained, might have been due to neuritis, but he had stated that he was unable to hold objects in his hand, yet found no difficulty in holding plate, spoon, knife and fork and using them freely. No signs of arsenical poisoning affecting other organs or systems had been present. It was probable that the patient exaggerated to a considerable extent any disability which he might have. The arsenic in the urine and fæces did not necessarily indicate poisoning by that substance.

Fusion of Nymphæ.

DR. R. I. FURBER, D.S.O., showed a child, aged six years. The mother had noticed that the vulva of the patient was abnormal in infancy, but no symptoms had developed until three months previously when the patient had complained of irritation. The *labia minora* had been found united almost completely and micturition had been effected through a hole close to the clitoris, the hole just allowed the passage of a probe. The fusion had given the impression that it was membranous only and that the irritation was due to the urine collecting above it. The *labia minora* had been easily separated by the knife and hymen and vagina had been found quite normal.

Dr. Furber stated that although the condition was not rare, the fusion in the patient he had shown, was carried much further towards the clitoris than in any figured in the textbooks.

A MEETING OF THE SYDNEY HOSPITAL CLINICAL SOCIETY was held in the Maitland Lecture Theatre, Sydney Hospital, on November 19, 1925, DR. T. FIASCHI, D.S.O., the President, in the chair.

Granuloma Tropicum.

DR. LANGLOH P. JOHNSTON showed a patient who had suffered from *granuloma tropicum*. The account of this case will be published at a later date.

Prostatic Stone.

DR. REGINALD BRIDGE showed a male patient, aged seventy-two years, who had complained of difficulty in passing urine for a period of seven months together with nocturnal frequency. The urine had been clear and free from pathological products. Residual urine amounting to sixty cubic centimetres (two fluid ounces) had been present. On rectal examination it had been discovered that the prostate was only moderately enlarged. About the centre of the left lobe there had been an area of stony hardness of the shape and size of a date seed. X ray examination had revealed a shadow corresponding in size and shape to the area of hardness in the left lobe of the prostate. At first sight the condition had appeared to be one of stone in the prostate.

A metal probe had been placed along the forefinger which had been inserted into the rectum. The tip of the finger had rested on the hard area of the prostate and a second skiagram had disclosed the shadow on the right side. When the patient was turned on to his left side and shaken, a third skiagram disclosed the shadow on the left side. Cystoscopy had also revealed a small stone in the bladder. It had therefore been determined that the patient was suffering from a stone in the bladder with a hard mass in the left lobe of the prostate suggestive of early carcinoma. Under local anæsthesia the bladder had been opened without the experience of any pain by the patient and the stone had been removed. The prostate had been enucleated under light chloroform anæsthesia. The patient, a frail old man, had made an excellent recovery. Examination of the left lobe of the prostate had revealed early carcinomatous change. The remainder of the prostate had presented the ordinary picture of senile hypertrophy.

Incontinence of Urine.

Dr. Bridge also showed a woman, aged twenty-nine years, who had been admitted to hospital with the history that she had suffered since childhood from mild incontinence of urine, especially on exertion. Small amounts of urine had frequently escaped from the bladder, but the patient was never inconvenienced by it. Three years prior to admission she had become pregnant and during the pregnancy the incontinence had become worse and remained so after the confinement. She had then submitted to some sort of plastic operation on the bladder neck and this had made her much worse. In addition, she had become affected by pyelonephritis on the right side and had been confined to bed for seven weeks suffering from intermittent attacks. Examination had revealed a short patulous urethra and absolute incontinence of urine. The urine had been foul smelling and had contained both pus and bacteria. She had found it necessary to wear numerous towels to keep herself dry.

On November 20, 1925, the patient had been submitted to cystoscopy and a most unusual condition was revealed. Dr. Bridge pointed out that in the female the ureteric orifices were quite often situated very close to the internal vesical sphincter; in fact in some instances they appeared to be situated on its margin. Any one familiar with the use of the cystoscope could verify this by direct observation. In this patient the orifices of the ureters had been situated actually beyond the sphincter of the bladder and opened into the urethra. This no doubt accounted for her life-long incontinence of mild degree. The compressor muscle of the urethra had been sufficient to prevent total escape of the urine. The plastic operation had apparently been carried out without the peculiar situation of the ureteric orifices being discovered and it had so damaged the compressor muscle that absolute incontinence had resulted and the right kidney had easily become infected. X ray examination had revealed the presence of two stones in the left kidney. They were producing no symptoms. The function of the right kidney as revealed by tests was very poor and pyelography had disclosed greatly dilated calyces, the kidney being only a shell.

Dr. Bridge said that this case showed the necessity for the cystoscopic examination of all patients suffering from incontinence of urine. Had this been done it would have been realized even in the absence of history that the incontinence which was really only partial, was due to an abnormal situation of both ureteric orifices in the urethra and that she was dependent on her *compressor urethræ* muscle to retain the urine in the bladder.

Sarcomata.

DR. GEORGE R. HAMILTON showed two patients. One, a youth, was suffering from sarcoma of the alveolar margin and the other was suffering from a fungating melanotic sarcoma of the right leg and the right temporal region. Dr. Hamilton asked for opinion in regard to treatment. It was generally agreed by those present that X ray treatment was the only possible means.

A Case for Diagnosis.

DR. GEORGE BELL, O.B.E., showed a patient and asked for suggestions as to the diagnosis. The patient, a female, aged thirty years, single, had been admitted to Sydney Hospital on May 4, 1925. Inquiring into the family history revealed that the father, aged eighty-four, was alive and well; the mother, aged sixty-nine, was alive; two brothers were living, one was not strong, had undergone twenty operations and had been cured by the removal of the appendix; of four sisters one was dead. The patient had complained of severe and continuous pain in the right lumbar region which passed round to the right iliac region. The pain became worse when the patient sat up. She had had it ever since a nephropexy had been done eight months previously, an ache before the operation had become a severe pain afterwards. The pain at times caused nausea. No pain accompanied micturition, but the patient passed urine six or seven times in the night and only twice during the day. The urine was sometimes cloudy; no blood had been passed. The appetite was good. There was no dyspepsia. The patient was extremely tender on palpation of the right lumbar region and was very tender on pressure over a small part of the scar immediately below the last rib (twelfth dorsal nerve).

On May 6, 1925, chromocystoscopy had been carried out. Indigo carmine had been excreted freely and urine stained with it had been seen issuing from both ureteric orifices. After pyelography in which a 25% solution of sodium bromide was used, Dr. Edwards had reported that some constriction of the ureter was present at its junction with the pelvis together with some dilatation of the uppermost calyx.

On May 13, 1925, ureteral catheters, sizes 7, 9 and 10, had been passed up the right ureter.

On May 21, 1925, after a consultation with Dr. Corlette, Dr. Bell had excised the last dorsal nerve from the cicatrix and also the ilio inguinal nerve under general anæsthesia.

Examination of the urine had been carried out. A culture made on May 13, 1925, had yielded Gram-negative motile bacilli. On May 19, 1925, epithelial cells, a few pus cells and a moderate number of red cells had been found. No casts were seen and culture yielded no growth.

The patient had been discharged from hospital on June 19, 1925, wearing an abdominal belt.

The pain had been unrelieved and as the patient complained of severe dysmenorrhœa she had been admitted under the care of Dr. George Armstrong. He had found that the uterus was retroverted and the right kidney collapsed, but not movable.

On September 10, 1925, abdominal section and curettage of the uterus had been performed by Dr. Armstrong. He had removed a long appendix, kinked in two places, and had performed ventrosuspension.

On October 1, 1925, the patient had still complained of attacks of pain on the right side, the pain being brought on when she sat up.

On October 28, 1925, two pyeloureterograms had been taken with the patient almost in the erect position and after she had drawn a deep breath. The constriction of the ureter seen on the previous occasion was apparently still present. The kidney had been found to lie opposite the third and fourth lumbar vertebræ. Dilatation of the renal pelvis had caused pain, but the patient had not appeared to be certain whether this pain was the same as that of which she usually complained. Subsequently a

further examination of the urine had been made and it had contained epithelial cells in abundance and a few pus cells. On November 1, 1925, the blood urea nitrogen had been eighteen milligrammes and the blood creatinine 2·5 milligrammes per hundred cubic centimetres.

Further X ray examinations had been made. A shadow had been seen at the left uretero-vesical junction and this was regarded as possibly being due to a calculus. A pronounced degree of gastroptosis had been found and Dr. Edwards had reported that the gall bladder contained some salts, but that its size was not extensively decreased.

On November 6, 1925, Dr. Holmes à Court had seen the patient. He had drawn attention to some doubtful scars about the knees. He thought that if the serum failed to react to the Wassermann test, if pain could be reproduced by dilatation of the renal pelvis and if the left renal function was normal, right nephrectomy should be performed.

Anterior Poliomyelitis of the Cerebellar Type.

DR. JOHN HUNTER showed a boy, aged six years, who had been admitted to hospital on October 16, 1925, with a history that while at school he had suffered from an attack of vomiting with sudden inability to walk. His previous history was unimportant. The onset of the illness had been sudden, no prodromal stage had been present and the boy had complained of neither pain nor diarrhœa. His temperature had been 37·2° C. (99° F.), his pulse rate 120 and his respirations 28 in the minute.

On examination of the cerebral nerves no abnormality had been detected. The *oculi fundi* had been normal, no papilloedema had been present. The function of the voluntary muscles of hands and arms had been normal, no spasticity or hypotonus had been found. The abdominal and epigastric reflexes had been normal. Examination of the leg had revealed no loss of power, no spasticity, absence of knee jerks and of ankle clonus, a flexor response to the plantar reflex, absence of Kernig's sign, normal sensation and absence of hyperæsthesia. The organic reflexes had been normal. The child had been very intelligent and his perception quick. Coordination in the arms had been good, but movement had been characterized by a coarse tremor. In regard to the legs the patient could neither walk nor stand (astasia abasia), but if lifted up he had been able to perform the walking movements with ataxia. No nystagmus had been present.

The spinal fluid had been examined, it was under slightly increased pressure, but was otherwise normal. No reaction had occurred to the globulin test, the Wassermann test and the gold sol test. The urine had contained neither albumin nor sugar.

Dr. Hunter said that the condition was diagnosed from syphilis by the absence of response to the Wassermann test and from tuberculous meningitis by the character of the spinal fluid and by the course of the disease. He said that Batten and Thursfield had described a cerebellar form of infantile paralysis with an unusual onset of headache, fever and vomiting. Ataxia quickly ensued and might affect arms or legs or both. It became evident on voluntary movement. If such a child were helped to stand up on his legs he might support the weight as long as his balance was maintained by the person holding him. If, however, the child was left to stand by himself, he at once assumed a wide base and if he attempted to walk, wild ataxia resulted. There was no loss of power and all movements could be performed in an ataxic manner. Intelligence was unaffected and nystagmus might be present.

Dr. Hunter said that the child was rapidly improving.[1]

Skiagrams.

DR. J. G. and DR. W. EDWARDS showed some interesting skiagrams. The lesions represented included osteo-arthritis, absence of the lung, multiple osteomata, stone in the bladder, spinal curvature, pneumothorax and hydro-pneumothorax.

[1] Dr. Hunter subsequently reported that one month after demonstration the child was walking fairly well.

THE MATER MISERICORDIÆ HOSPITAL, BRISBANE.

A CLINICAL MEETING of the Staff was held at the Mater Misericordiæ Public Hospital, Brisbane, on November 18, 1925, DR. E. D. AHERN in the chair.

Ocular Injury.

DR. ALEX. MACDONALD showed a blacksmith who, whilst striking a piece of hot metal on an anvil, had received an injury to his right eye. A small piece of metal had flown up and perforated both the cornea and the sclera and opened up both chambers of the eyeball. All the aqueous and most of the vitreous had flowed from the wound. The piece of metal luckily had been sterile on account of the heat and the wound was not infected. The patient had been put to bed and the wound cleaned under local anæsthesia, a new anterior chamber formed and the eye had become quite healed. There was present an anterior synechia with adhesions of the iris to the back of the cornea. By means of pressure a flat scar was obtained in the cornea. The patient's vision was $^{2}/_{60}$ and the intra-ocular pressure was good.

The second patient shown by Dr. MacDonald was a boy, also employed in a blacksmith's shop. His injury had been produced in the same manner by a piece of metal flying up from the anvil and injuring the cornea and the lens. The wound had completely healed. There was a cataract forming in the injured lens. A new anterior chamber had developed and there was good pressure in the eyeball. Dr. MacDonald thought that later the cataract could be needled and the vision of the injured eye greatly improved.

Tumour of the Neck.

DR. ELLIS MURPHY demonstrated a tumour the size of a small orange in the posterior triangle of the neck. The patient was a girl, aged twenty-one years and had first complained of the swelling in her neck eighteen months previously. During the previous twelve months she had received numerous doses of X rays to the mass which became a little larger after each application and settled down a short time afterwards. At the time of demonstration the tumour was bigger than ever. It consisted of numbers of enlarged lymph glands which were more or less discrete, the skin was movable over the tumour and the tumour itself was movable on the underlying structures. Dr. Murphy thought the condition was one of lymphadenoma.

DR. B. L. W. CLARKE described the X ray treatment to which the patient had submitted and said that in his opinion the patient was suffering from lymphadenoma.

DR. E. S. MEYERS and DR. L. M. MCKILLOP thought that the tumour should be removed.

DR. NEVILLE SUTTON described the pathological findings in Hodgkin's disease and lymphadenoma.

Parkinson's Syndrome.

Dr. Murphy next showed a case of post-encephalitic bradykinesia or Parkinson's syndrome. The patient was a woman, aged forty years. Up to three years previously she had been a bright happy woman, noted for the great amount of work she could do. At this time she had a severe attack of influenza during which she was laid up for a fortnight and suffered very severe pains in the back of her neck, her back and in her limbs. Three months later she noticed that her hands became tremulous and she began to lose interest in her surroundings. The condition had gradually progressed to her present state. The patient manifested a definite Parkinsonian mask, the face being immobile and devoid of the natural play of expressions. She walked in a stooped attitude with her elbows pressed to her sides and the forearms slightly flexed on the arms. The gait was shuffling, when she turned, her head moved with her body as if fixed. The sternal attachments of the sterno-mastoid muscles were prominent. The ordinary nictitating movements of the eyelids were diminished. The fingers were very tremulous, though there were no definite "pill rolling" movements. In spite of the great tremor which appeared to be increased with volun-

tary movement, she could write her name and address quite well. Her mentality was not affected, her knee jerks were present and a little exaggerated, as were her ankle jerks. The plantar reflex was flexor and there was no abnormality of sensation. The pupils reacted to light and to accommodation. Dr. Murphy considered the condition to be due to damage to the brain caused at the time of "influenza" when the patient probably suffered from a mild attack of encephalitis.

DR. P. J. KELLY stated that he had seen several patients with *encephalitis lethargica* who had afterwards developed symptoms identical with this patient.

DR. NEVILLE SUTTON had also seen a boy with encephalitis who had later developed a mask-like face with some slowness of movement, but no tremor.

DR. ELLIS MURPHY said that the French writers made a definite distinction between post-encephalitic bradykinesia and *paralysis agitans*. In both the seat of the lesion was in the *globus pallidus* of the lenticular nucleus. The most important difference lay in the prognosis; patients with *paralysis agitans* tended gradually to go down hill to total paralysis and death, whereas the post-encephalitic patients remained stationary or tended to get better.

Tabes Dorsalis.

The third patient shown by Dr. Murphy was suffering from *tabes dorsalis*. The patient was a man aged forty-three years. Four months previously he had sought treatment for acute pains in his right knee with swelling of the joint. The pains had been burning in character and lasted several minutes at a time and had been accompanied by synovitis of the knee joint with increased synovial fluid. One month previously the patient began to get similar pains in the left knee and during the last two weeks he had complained of a numb feeling across his chest and some slight staggering when walking. More than four months previously he had had many attacks of "rheumatism" in his knee. The patient had a definite steppage gait, he lifted his feet higher than necessary and planked them down with unnecessary violence. His pupils were small and a little irregular in outline, the right being larger than the left. They reacted only very slightly to a strong light and very well to accommodation. His knee and ankle jerks were absent, there was no plantar reflex. Squeezing of his calf and thigh muscles caused no pain. There was definite Rombergism. Sensation was delayed, though there was no definite analgesia of the skin of the nose or around the anus. There was no wasting or loss of power in the muscles. According to the patient his sight was good. He had difficulty in walking down stairs. The response to the Wassermann test was ++. Dr. Murphy laid stress on the necessity for the diagnosis of *tabes dorsalis* in the early stages. The "lightning" pains frequently appeared months or even years before the classical symptoms of Argyll-Robertson pupil and loss of knee jerks. Many of the so-called attacks of "rheumatism" were in reality "lightning" pains. The ankle jerks often disappeared months before the knee jerks were affected. Strong antisyphilitic treatment should be used as soon as possible.

Urological Examination in Diagnosis.

DR. A. S. ROE showed a female patient in whom he demonstrated the value of urological examination in diagnosis. The patient had been sent to him from the surgical ward for investigation on account of a large tumour in the left hypogastrium, for diagnosis between enlargement of the spleen and left kidney. The cystoscope had revealed the signs of acute cystitis. The indigo carmine test had been performed and in six minutes the dye was seen issuing from the right ureteral orifice. Twenty minutes later no appearance of the dye on the left side had been noted. The ureteral catheter had been passed up to the pelvis of the left kidney and fifteen cubic centimetres of solution had been injected without pain; it had even flowed down the sides of the catheter. A catheter specimen had contained bacteria and pus, thus demonstrating the presence of pyelo-nephrosis and showing that the tumour was due to the kidney.

Prostatic Enlargement.

Dr. Roe showed several specimens of prostates and demonstrated the various enlargements which could occur.

Cystitis.

Dr. Roe then described the case of an old lady who periodically suffered from very acute attacks of cystitis which failed to respond to all ordinary methods of treatment. This patient had received twenty cubic centimetres of "Mercurochrome," representing five milligrammes per kilogram of body weight. The injection had been made intravenously. There had been a very severe general reaction. Fourteen days later the cystoscope had revealed great improvement in the condition of the bladder wall. The mucous membrane had been almost clear and the blood vessels were visible. Dr. Roe said that he would not care to say that the cure was permanent.

Diathermy.

DR. B. L. W. CLARKE showed a man who had received diathermy treatment for an epithelioma of the soft palate in November, 1921. The patient had had three applications of diathermy and one of deep X rays. Some weeks previously he had lost the power of swallowing.

Abdominal Injury.

DR. E. D. AHERN showed a young man who had been seriously injured two years previously. He had been thrown from a horse and sustained severe injuries to the abdominal wall and perineum and had been treated for many months in a country hospital. On admission to the Mater Misericordiæ Hospital the lower part of his anterior abdominal wall had been absent, the inside of his bladder had been exposed and the ureteral orifices could be seen. He had developed an acute pyelo-nephrosis which had been evacuated through the rectum. A colostomy had been performed. The patient had made great headway since receiving intravenous injections of gentian violet. The wound in the abdominal wall had almost healed, he could retain three hundred and sixty cubic centimetres (twelve ounces) of urine and he had put on 7·2 kilograms (sixteen pounds) weight in the previous six weeks.

Empyema.

Dr. Ahern also showed a girl whose condition had been diagnosed as pulmonary tuberculosis. She had spent two years at the Dalby Sanitarium and eight months at Diamantina Hospital for Incurables. He had demonstrated the presence of empyema which had tracked along certain of the ribs, the cavity had been laid open and drained and the patient had received intravenous treatment with a solution of gentian violet. She had improved greatly since operation.

Branchial Cyst.

Dr. Ahern's third patient was a woman from whom a few days previously he had removed a branchial cyst. The tumour had been as large as a cricket ball and its base had been traced in between the mastoid and the styloid processes, the site of the second branchial cleft. The patient had had a small tumour in the region for many years, but when she became pregnant it immediately began to enlarge, increasing in size from that of a hazel nut to its size before operation.

Follicular Conjunctivitis.

DR. WALLIS HOARE showed a boy suffering from follicular conjunctivitis and demonstrated the translucent swelling of the conjunctival membrane. He explained the differences between this condition and trachoma. Follicular conjunctivitis was found only in children, especially those suffering from adenoid hypertrophy and was common in schools.

Pathological Specimens.

DR. L. M. MCKILLOP showed the following specimens:

Dermoid cyst removed from the thigh; malignant tumour removed from the neck, stated by the pathologist to look

rather like psammoma; myeloid sarcoma of lower jaw; ovarian calculi; ovarian cyst and fibroid tumour of the broad ligament from the same patient.

THE ALFRED HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE ALFRED HOSPITAL CLINICAL SOCIETY was held at the Alfred Hospital on February 23, 1925, DR. M. D. SILBERBERG, the President, in the chair.

Injury to the Forearm.

MR. H. C. COLVILLE showed a girl, aged seven years, who in mid-December, 1925, had been knocked down and dragged along the ground by a lorry. On admission to hospital she had been treated by immediate operation, the wound being cleaned and hopelessly damaged tissue being excised. The elbow joint had been opened and cleaned out. The head of the radius had been replaced and the capsule of the joint sutured. The whole of the skin and muscles, indeed all of the tissues on the dorsum of the forearm down to the bones and the interosseous membrane had been torn away. The tendons of the extensor muscles had been cut just above the wrist joint. The *supinator longus* and the *extensor carpi ulnaris* muscles alone remained. Healing had progressed until on January 28 the raw area left was grafted by Thiersch's method. At the time of demonstration the wound surface was healed. The girl could extend the fingers feebly by using the interossei and lumbrical muscles, but extension at the wrist joint was absent. The function of the elbow joint was moderately good. Dr. Colville asked for suggestions as to further treatment.

MR. HAMILTON RUSSELL congratulated Mr. Colville on his handling of the case. He suggested that possibly later on tendon transplantation should be tried. Function, he said, should be good so long as dorsiflexion at the wrist joint was maintained.

MR. J. KENNEDY suggested that an attempt be made to effect an arthrodesis of the wrist joint. The operation would be difficult but, he thought, possible.

MR. BALCOMBE QUICK suggested that the remains of the tendons of the extensor muscles of the wrist should be fixed into the radius, so to act as ties keeping the joint in the required position.

Chronic Lymphatic Leuchæmia.

DR. M. D. SILBERBERG showed a woman, aged forty-nine years, who was suffering from chronic lymphatic leuchæmia. At the onset about nine months previously the first symptoms had been weakness and pallor, dyspnœa and palpitation, anorexia and nausea with occasional vomiting. Four months previously she had first noticed a left-sided abdominal tumour which had become very large. There was no pain but only a dragging sensation caused by it.

Enlarged glands had appeared about three months previously. In the last two months giddiness and weakness had become much more prominent. She had noticed numbness and tingling in the feet.

On examination pallor was present, there was an enormous enlargement of the spleen which extended downwards into the pelvis and across the midline at the umbilicus for about five centimetres (two inches). It was smooth and gave rise to no pain. All the superficial lymph glands were moderately enlarged. They were smooth, discrete and elastic. The liver was slightly enlarged. The leucocyte count was 600,000 per cubic millimetre and 92% of the cells proved to be small lymphocytes. The erythrocyte count was 2,000,000. The hæmoglobin estimation was 40% and the colour index one. There was slight anisocytosis and poikilocytosis. No abnormality was to be detected in the nervous system or in the lungs or in the fundi. The heart was slightly enlarged and faint bruits were heard, apparently they were functional in character. The urine contained no albumin or sugar. Fouchet's test yielded no response. Examination of the stomach contents revealed an achlorhydria. A skiagram of the chest presented no abnormality.

In regard to treatment Dr. Silberberg said that it was proposed to use benzol for a short period. Already the white cells had fallen to 302,000 after the first few doses. Later the spleen, glands and perhaps the long bones would be irradiated.

DR. SPALDING LAURIE stated that he had had under observation a similar patient for the previous six or seven years. The enlargement of spleen and glands was similar. X ray treatment repeated frequently, at times as often as once a month, had had a beneficial effect and had kept down the white cell count.

DR. J. F. MACKEDDIE, commenting on the absence of free hydrochloric acid in the stomach contents, remarked that this symptom was common in anæmia. He had seen it in cases of familial acholuric jaundice and in Banti's disease. When recently in England he had met many who considered such a symptom a rarity, but he could assure the members of the Society that it was not so.

Lesion of Nervous System Affecting the Arms.

DR. G. A. BIRNIE showed a woman, aged forty-six years, who had complained of a loss of power of both hands for the previous ten weeks. She woke in the morning with sensations of numbness and tingling in both hands and fingers. The condition had steadily and progressively become worse. On examination a symmetrical bilateral wasting of the thenar eminence was found and no other wasting. There was a patchy anæsthesia over both arms and forearms, but no other abnormality of the nervous system. All other general examination failed to reveal further abnormalities. X ray examination revealed on each side a moderately large cervical rib. Dr. Birnie said that he felt confindent that the cervical ribs were not the cause of her symptoms.

DR. J. F. MACKEDDIE agreed that the cervical ribs were not the cause of the trouble. Two points were of interest in the case. As a rule in a peripheral lesion the wasting was out of proportion to the weakness; that was not so in the case under discussion. On examination of the patient he had elicited the fact that there was also some loss of power of the deltoids and of the forearm. There was also in his opinion some evidence of ataxia. He considered that the syndrome presented was due to a combined sclerosis, a posterior column degeneration essentially of central origin.

It was true that in cervical rib cases one frequently found a wasting of the thenar eminence. Kinnear Wilson held that that wasting was due to the supply of the muscle by fragments from the eighth cervical through the median nerve. Still in this case, as he said, he considered the cervical ribs to have no bearing on the diagnosis.

DR. W. SUMMONS remarked that the case presented many of the features of an ordinary peripheral neuritis. He too was of the opinion that the cervical ribs were not the cause of the symptoms.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING OF THE SENATE OF THE UNIVERSITY OF SYDNEY was held on April 19, 1926.

Mr. A. C. McKay, B.V.Sc., was reappointed to the Walter and Eliza Hall Veterinary Science Fellowship for a period of three months.

The following appointments were made:

Dr. D. W. McCredie as Acting Tutor in Surgery at the Royal Prince Alfred Hospital.

Drs. M. R. Flynn, D. W. McCredie, T. H. Small and C. L. Chapman as Honorary Demonstrators in Anatomy.

Dr. J. L. McKelvey as Lecturer in Clinical Surgery at the Royal Prince Alfred Hospital.

Dr. B. T. Edye as Lecturer in Clinical Surgery at St. Vincent's Hospital.

Mr. C. G. Gostelow as full-time Demonstrator in Pharmacy and Mr. K. E. Hinton as part-time Demonstrator in Pharmacy.

Mr. J. Bannon as Demonstrator in Physics, and Dr. T. Cooksey as part-time Demonstrator in Physiology.

The Dean of the Faculty of Medicine, and Drs. Purser, Abbott, Blackburn and D'Arcy were appointed as representatives of the Senate to act with five members of the Board of Directors of the Children's Hospital as a Conjoint Board.

Messrs. George Wright and H. E. Ross were appointed as members of the Cancer Research Committee.

The following by-laws governing the curriculum for medical students were approved:

By-Laws.—Chapter XII., Sections 4 and 5.

Section 4.—Candidates for the Degree of Bachelor of Medicine and Bachelor of Surgery shall attend the following courses of instruction:

I. In the First Year—
- Botany, including Laboratory Practice—One term.
- Chemistry (Inorganic and Organic), including Laboratory Practice—Three terms.
- Physics, including Laboratory Practice—Three terms.
- Zoology, including Laboratory Practice—50 Lectures and 72 hours Laboratory Practice.

II. In the Second Year—
- Anatomy—Three terms.
- Dissections—Three terms.
- Practical Histology—Eighty hours.
- Physiology, including Laboratory Practice—Three terms.

III. In the Third Year—
- Dissections—Three terms.
- Anatomy, including Applied Anatomy—Three terms.
- Physiology, including Laboratory Practice—Three terms.

IV. In the Fourth Year—
- Surgery—Lent and Michaelmas terms.
- Pathology and Bacteriology—Three terms.
- Operative Surgery—Lent term.
- Medicine—Michaelmas term.
- Hospital Practice, including Tutorial Surgery, Tutorial Medicine, Clinical Surgery, Surgical Out-patients daily, including Saturdays—Three terms.

V. In the Fifth Year—
- Medicine—Lent and Trinity terms.
- Obstetrics—Lent term.
- Surgery—Trinity term.
- Therapeutics and *Materia Medica*—Michaelmas term.
- Gynæcology—30 lectures, Trinity term.
- Medical Jurisprudence—20 lectures, Trinity term.
- Preventive Medicine—50 lectures, Michaelmas term.
- Hospital Practice, including Clinical Surgery, Clinical Medicine—Three terms.
- Practical Pharmacy—Twenty hours.
- Pharmacology.
- Clinical Obstetrics—Nine weeks.

VI. In the Sixth Year—
- Diseases of the Skin—Ten lectures with demonstrations.
- Diseases of the Ear, Nose and Throat—Ten lectures with demonstrations.
- Psychiatry—Thirty lectures, including demonstrations.
- Diseases of the Eye—Ten lectures with demonstrations.
- Medical Ethics—Three lectures.
- Clinical Pathology—in continuous groups (6 weeks).
- Clinical Biochemistry—in continuous groups (6 weeks).
- Hospital Practice, including Clinical Medicine, Clinical Surgery—Daily.
- Children's Hospital—6 weeks continuous course.
- Clinical Obstetrics—Refresher Course—Two weeks.

Section 5.—For the Degrees of M.B. and B.S. the examinations shall be as follows:

(i.) A First Degree Examination at the end of the First Year in Chemistry, Physics, Zoology and Botany.

(ii.) A Second Degree Examination at the end of the Second Year in Anatomy and Physiology.

(iii.) A Third Degree Examination at the end of the Third Year in the entire subjects of Anatomy (including Applied Anatomy) and Physiology.

No candidate shall be admitted to this Examination unless he has completed the dissection of every part of the body at least once.

(iv.) A Fourth Degree Examination at the end of the Fourth Year in Pathology.

(v.) A Fifth Degree Examination at the end of the Fifth Year in Medical Jurisprudence and Preventive Medicine and Pharmacology, Therapeutics and *Materia Medica*.

(vi.) A Final Degree Examination at the end of the Sixth Year in Medicine, including Clinical Medicine; in Surgery, including Clinical Surgery and Operative Surgery; in Obstetrics, including Clinical Obstetrics and Gynæcology; and in the subject-matter of the lectures of the Sixth Year.

Hospitals.

HOSPITALS FOR THE INSANE IN VICTORIA.

THE annual report of the Inspector-General of the Insane of Victoria for 1924 which was issued early this year, provides a considerable amount of interesting matter. The form of report is the same as in previous years. Unfortunately it has been impossible in recent years to discuss these reports owing to the large amount of material awaiting publication and the small space available in the weekly issues.

Statistics.

The total number of persons certified as insane in the State of Victoria on January 1, 1924, was 6,016. During the course of the year this number was increased by 809 persons admitted to the various institutions and reduced by 723, representing 293 patients discharged and 430 patients who died, as well as sixteen patients who escaped and were not recaptured. The total number on December 31, 1924, was therefore 6,096. The number of persons under certificate is actually ninety-five more, this representing the number of persons committed to private licensed houses. In addition to the total of 6,191, 267 persons were under treatment without certification. The ratio of the insane to the population was 1: 268. We venture to point out that it is unsafe to accept these figures without considerable reservation. Diseases of the mind like diseases of other functions or organs exist in mild or atypical forms and these cases remain outside the official recognition. In addition some people with diseases like paranoia or delusional insanity remain at large without medical treatment. It would be safer to state that in every two hundred and sixty-eight persons one is known to be insane, but that it is highly improbable that the remaining two hundred and sixty-seven are all sane.

Of the 809 persons admitted during the year, 663 had not previously been in a hospital for the insane. There were one hundred and seventy-nine persons discharged under the rubric "recovered" and one hundred and one discharged under the rubric "relieved." It appears that while between two and three hundred patients are discharged each year, about one hundred and fifty are subsequently readmitted. Again there is reason to question whether the majority of those said to have recovered, can be regarded as having been permanently freed from all traces of their mental disease. The calculation of the ratio of those who recover

to those who are admitted, appears to us to be a very arbitrary measure. This method does not disclose the prognosis of insanity.

The death rate among the inmates of the hospitals has remained remarkably constant during the past quarter of a century. It is higher among the males than among the females. This fact may to some extent account for the higher mean age of the female than of the male patients. The death rate of males exceeded 10% of the average number resident on but two occasions. In one year, namely 1906, it was as low as 6·71%. With these three exceptions the death rates of male patients have varied between 8% and 10% with an average of 9·19%. In 1910 and in 1922 the death rate of female patients was below 6% (5·85% and 5·32%). In all other years it varied between 6% and 7·93% with an average of 6·71%. Of the 430 deaths, no less than eighty-nine were caused by pneumonia, bronchitis and pleurisy, fifty-two were due to old age, forty to diseases of the heart, forty to general paralysis of the insane, twenty-six to pulmonary tuberculosis, twenty-five to diseases of the genito-urinary system and twenty-four to malignant disease elsewhere than in the brain. Relatively few deaths were directly caused by the cerebral disease.

From the tables it appears that 934 of the 6,096 persons remaining on the lists at the end of the year, or 15·3%, were suffering from congenital or infantile mental deficiency occurring as early in life as can be ascertained, while of the remainder primary, senile and secondary dementia accounted for the illness of 3,098 or 60% of those whose insanity occurred later in life. There were 200 patients with insanity with epilepsy, 121 with chronic mania, 111 with systematized and 432 with non-systematized delusional insanity and 178 with recent, chronic or recurrent melancholia. The number of general paralytics was forty-six.

Pathological Work.

The Inspector-General calls special attention to the burden placed on the shoulders of the pathologist who is required to make a *post mortem* examination of every patient who dies in the metropolitan institutions. Dr. Ernest Jones holds that these examinations are devoid of scientific value in a large number of instances because the patients have died from senility. We are not prepared to endorse this opinion, for every autopsy may be of considerable scientific importance, no matter what the actual cause of death may be, provided that the pathologist has sufficient time to study the many abnormal conditions encountered in a dead body. It must be admitted, however, that it is extravagant and wasteful to require a skilled pathologist to spend a great deal of time in making examinations after death and in giving evidence before a coroner. There is no reason why many of these examinations should not be conducted by the medical officers of the hospitals. If this were done, Dr. W. A. T. Lind would have time to pursue his investigational work. Reference is made in the report to his articles published in this journal on the causation and pathology of Mongolian idiocy and epiloia and on perforated ulcer of the ilium.

The Psychiatric Clinic.

The Inspector-General again introduces the question of the care and treatment of patients suffering from psychoses in their early stages without the restraint of certification. Attempts have been made on numerous occasions to induce the managers of the general public hospitals to equip psychiatric wards and out-patient departments. Although this advice has not been followed in its entirety, something has been done in this direction. Dr. Morris Gamble has been appointed Consulting Alienist to the Melbourne and the Alfred Hospitals. At the Alfred Hospital an out-patient department has been organized. Provision has already been made for the reception of voluntary patients at some of the institutions under the control of the department. But it is eminently desirable that neurotic patients and those with early mental changes should be able to receive curative treatment in general hospitals without any association with hospitals for the insane. Moreover it would be a great advantage to medical students if they could receive instruction in the diagnosis and treatment of mental disease in its earliest stages in the hospitals attached to the medical schools.

NEW INSTRUMENTS.

THE Labat Outfit for spinal or regional anæsthesia has gained many friends in other parts of the world and will doubtlessly become popular in Australia. It comprises two syringes, one of two cubic centimetres capacity, made in two parts of glass, and one of twenty cubic centimetres capacity, with a glass barrel and metal plunger and nozzle. The nozzle in the larger syringe is eccentrically placed and the needles are fitted by means of a bayonette lock. The metal case contains a convenient tray with a suitable handle, so that the whole outfit can be sterilized on it without the need of forceps to remove each portion. The instruments are well made, handy and of reasonable price. The one defect of the all-glass syringe, namely the fragility of the nozzle piece, is obviated in the larger syringe, while the serious defect of the "Record" type of syringe is reduced to a minimum by the excellent fit of the massive metal plunger. The needles are of varying sizes and are carefully made. The outfits are obtainable from the agents of the Anglo-French Drug Company, Limited, Messrs. Maclay Brothers, B.M.A. Buildings, 30 to 34, Elizabeth Street, Sydney.

Correspondence.

A WARNING.

SIR: The following case may act as a warning to some of those middle aged or elderly practitioners who find themselves losing their pristine enthusiasm and getting into the dubious habit of "leaving it to Nature." The writer was called to see "M.D.," a Swede, aged seventy-five years, at 6 p.m. one evening. The patient was found in bed in a small room in a small cottage with a large hard painful inguinal hernia which when carefully palpated showed all the signs of acute obstruction, if not strangulation. No fluid nor flatus could be recognized in the tumour which was tense and immovable. The patient had had no bowel movement for twenty-four hours and was retching intermittently. His pulse was slow and he looked ill and said he "could not lie down for the pain." I urged immediate operation at a nearby hospital and explained in gruesome detail the inevitable results of delay both to the patient and his wife and relatives. Everybody agreed with me except the patient who absolutely refused to have any operation or even to be removed to hospital. He said he was "sure it would go back" and that he would "wait till the morning." All preparations were made for immediate operation in case he should think better of it at any moment and after an hour's fruitless argument I retired for the night. At seven o'clock next morning I visited the patient and found him quite comfortable. The hernia had gone back.

Yours, etc.,

"DR. NIL."

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned has been registered under the provisions of the *Medical Act*, 1912 and 1915, as a duly qualified medical practitioner:

Dearberg, John Louis, L.R.C.P. (Edinburgh), 1926, L.R.C.S. (Edinburgh), 1926, L.R.F.P.S. (Glasgow), 1926, 83, Oberon Street, Coogee.

For Additional Registration:

Petherbridge, Walter Charles, Ch.M., 1910 (Univ. Sydney), 55, Harrow Road, Bexley.

QUEENSLAND.

THE undermentioned have been registered, under the provisions of *The Medical Act* of 1925, as duly qualified medical practitioners:

Johnson, Horace William, M.B., Ch.M., 1925 (Univ. Sydney), Brisbane.

Power, Harold Horne, M.B., Ch.M., 1925 (Univ. Sydney), Brisbane.

Lee, Alan Edward, M.B., B.S., 1920, M.D., 1922 (Univ. Melbourne), F.R.C.S. (England), 1924, Brisbane.

Additional Qualification.

Murray, Gerald Aubrey, D.P.H. (Univ. Sydney), 1925.

Medical Appointments.

Dr. Leslie Scott Latham (B.M.A.) has been appointed Acting Official Visitor to Metropolitan Hospitals for the Insane, Victoria.

* * * *

The undermentioned have been appointed Members of the Midwives Board, Victoria: Dr. Edward Robertson (B.M.A.) (Chairman), Dr. Jane Stocks Greig (B.M.A.), Dr. William Ernest Jones (B.M.A.) and Dr. Clarence George Godfrey (B.M.A.).

* * * *

Dr. John McKee (B.M.A.) has been appointed Government Medical Officer at Bega, New South Wales.

* * * *

Sir Alexander MacCormick (B.M.A.) has been appointed a Director of the Australian Mutual Provident Society.

* * * *

Dr. Bronte Smeaton (B.M.A.) has been appointed a Member of the Advisory Committee under the provisions of the *Hospitals Act Amendment Act*, 1921, South Australia.

* * * *

Dr. Harold Mitchell Rees (B.M.A.) has been appointed an Honorary Medical Officer to the Port Pirie Hospital, South Australia.

Books Received.

CATALOGUE OF EARLY MEDICAL WORKS AND BIOGRAPHY. Issued by H. K. Lewis and Company, Limited, London. Demy 8vo., pp. 44. Gratis and post free to any address.

CATALOGUE OF EARLY SCIENTIFIC WORKS AND BIOGRAPHIES. Issued by H. K. Lewis and Company, Limited. Demy 8vo., pp. 12. Gratis and post free to any address.

THROUGH A CONSULTING ROOM WINDOW: BEING LITERARY AND OTHER ESSAYS FROM A MEDICAL HAND, by W. C. Rivers; with an introduction by Arthur Ponsonby, M.P.; 1926. London: Methuen and Company, Limited. Crown 8vo., pp. 211. Price: 6s. net.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xxii.

QUEEN VICTORIA HOSPITAL FOR WOMEN AND CHILDREN, MINT PLACE, MELBOURNE: Medical Superintendent, Senior Resident Medical Officer.

THE PUBLIC SERVICE BOARD, NEW SOUTH WALES: Assistant Medical Officer (Female).

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C. 1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

MAY 11.—Tasmanian Branch, B.M.A.: Branch.
MAY 13.—Victorian Branch, B.M.A.: Council.
MAY 13.—New South Wales Branch, B.M.A.: Clinical Meeting.
MAY 14.—Queensland Branch, B.M.A.: Council.
MAY 17.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAY 18.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 18.—Tasmanian Branch, B.M.A.: Council.
MAY 19.—Western Australian Branch, B.M.A.: Branch.
MAY 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 25.—Illawarra Medical Association, New South Wales.
MAY 26.—Victorian Branch, B.M.A.: Council.
MAY 27.—New South Wales Branch, B.M.A.: Branch.
MAY 27.—South Australian Branch, B.M.A.: Listerian Oration.
MAY 28.—South Australian Branch, B.M.A.: Branch (Annual).
MAY 28.—Queensland Branch, B.M.A.: Council.
JUNE 1.—Tasmanian Branch, B.M.A.: Council.
JUNE 2.—Victorian Branch, B.M.A.: Branch.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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